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Editor: H. A. Shapiro, Ph.D., M.B., Ch.B., F.R.S.S.Af.

Brain Waves

In recent years electrical discharges from the brain have been tapped and recorded as waves. This brain wave pattern is quite different from that observed in other organs such as the heart, the retina and the muscles, but disturbances in the normal pattern throw suggestive light on disturbances in the function of the brain—a situation analogous to what has been worked out for the heart.

Careful studies have revealed increasing certainty about the normal range of variation of brain wave patterns in children and adults; and how age, *inter alia*, affects the brain wave pattern. On this basis standards for the diagnosis of maturity are beginning to suggest themselves. Thus, under controlled conditions of testing, the brain wave pattern may provide reliable, objective and acceptable evidence of immaturity and instability and so throw light on the degree of responsibility of accused persons.

The study of brain wave patterns in epileptics and in certain types of murderer has already proved most rewarding and helpful in the administration of justice. Current studies are testing its value in the difficult field of the psychopath—in whom a case for diminished responsibility for his acts can, in certain circumstances, be made out.

New light is also being shed on the immaturity of delinquents. Brain wave studies have pointed to the possibly special significance of bed-wetting and fire-raising as elements in the complicated syndrome that makes up delinquency.

Clearly there are many ways in which these new studies will help us to understand anti-social behaviour. The forensic implications of this novel way of looking at the brain are very considerable. The recording and interpretation of brain wave patterns is a matter for the technical expert, but with the increasing use of this type of information in our courts there is a growing need for some familiarity with the new kind of language which the expert witness must use.

We therefore welcome the opportunity of publishing in this issue the first of two articles by Dr. A. C. Mundy-Castle who has written them as simply as technicalities and accuracy permit. From the very nature of the subject it cannot be presented in entirely lay terms. But the average and interested legal or medical reader now has available a reasonably intelligible introduction to a field of study which harbours a great fascination in its own right and which will yield invaluable assistance in settling some of the most complex problems that present themselves in our Courts and in our Society.

Psychiatric Problems of Offenders*

Lewis J. Siegal, M.D., LL.B.†
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The control of social life has developed upon historic rather than scientific principles and problems of behavioural deviation were dealt with during antiquity on a traditional instead of on scientifically evolved conclusions.

Medical science has had its development in a similar manner, and until a few hundred years ago, any physician who departed from what the books said had been done for centuries before, was regarded as an unorthodox medical practitioner. Then followed the era of scientific handling of problems of physical disease with its unending progression of improved results of which we are now startlingly aware almost daily.

Accordingly, the logical inference would be, that for another kind of human problem, namely crime—a similar change in method from historic to scientific, might be expected to yield equally gratifying results. It may be objected to, at once, by some, that crimes bear no relationship to, nor can they be concomitants of illness. To carry this further, they may attempt to postulate as a basis for such reasoning, that what one's organs may do, because of their endowment with specific physiological function, might well be a medical problem, but what one's limbs may do, in so far as they touch the rest of society—is a legal problem.

Because of this kind of reasoning it has not been until comparatively recently that conduct and behaviour have come to be regarded as governed by scientific laws. A study of the crime problem thereupon received impetus, and under the advantage of trained and scientific attitudes of objectivity, with its utter detachment from sympathy *per se* (whether for or against the offender), a method of approach has developed which is precisely opposite to that of the position of the prosecutor.

Scientists frankly avow the aim of protecting society, rather than achieving justice; to that end, from the standpoint of assisting society and reducing the menace of crime, psychiatrists (aided by the inestimable work of those engaged in psychology, social work and the indispensable and signal service of probation officials) aim only at the ideal, to wit: the achievement of protecting society from individuals incapable of living safely within it.

* Based on an address delivered at the New York Criminal Court, before the Probation-Officer Staff of the Court of Special Sessions, on 30 October 1953.
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As physicians we never talk of the justice of a medical case. We are never concerned whether the treatment of a case is just and unprejudiced treatment, but are concerned whether it is treatment that is expedient and beneficial. By this same token, as psychiatrists, we recognize that crime is symptomatic of disease, and specifically so treat it.

At one time all fever was treated alike and regarded as a disease. But to-day it is well recognized within all strata of society, even to the most uninitiated, that there are many kinds of fevers, and that they must be treated in many different ways, conformable to the specific illness.

Similarly, time will come when stealing and homicide will also be understood in the symptom category indicating presence of a disease—a personality disease—which may be one of a dozen or more different sorts, requiring any one of a score of differing therapeutic approaches.

In the long ago, the legal precept provided that anti-social behaviour which seemed to yield immediate profit or advantage to the participator should be called crime, and should be followed by officially inflicted revenge called punishment. Some will be heard to criticize punishments as being too severe; others will say that they are not severe enough. Still others will insist that although sufficiently severe, the punishment meted out is not certain enough. But all of these unscientific and opinionated authorities are agreed that if sufficiently fearful retributions could be devised, the offenders will stop offending! This is truly archaic.

The idea that back of such antisocial behaviour exists a complex, laden with physical, chemical and biological ingredients which are infinitely more painful than any blundering punishment, does not appear to penetrate these sentimentalists and theorists. The idea that a person may commit a crime without having the slightest conscious idea of why he does it, appears to be equally misunderstood by these proponents. The fact that an offender, with brain syphilis, is more likely to respond to our modern antibiotic therapy rather than to a term of years in prison, or that a moronic individual involved in a sexual crime is unable to adapt himself to society without its assistance—seems to lack the consideration of prosecutors, who would cure such and other assorted crimes by varying periods of imprisonment, to say nothing of some sentimentalists who would save souls of malefactors by rescue missions. Such correctives are an attack of the problem of crime on an emotional basis—hard or soft—rather than on a foundation of intellect and scientific handling within the climate of psychiatry and its collateral implements of psychology, social service and the indispensable protective sympathetic guardianship and supervisory social approach of probational superintendence.

In a general way, tendencies to criminal acts may be predicated to a minor extent upon qualities inherent in the offender's personal make-up and, to a greater degree, upon compelling situations arising within his social sphere. I have found that by far the greater percentage of criminality

and delinquency shows up in persons within the so-called normal population pyramid. In other words, these offenders are neither psychotic, neurotic nor mentally defective, but are experiencing, in the main, varying gradations of pressure-producing personality problems.

From my consultative contact with youthful offenders, as well as delinquents processed through the Children's Court, I have come to conclude that their entanglements with the law are predominantly founded upon feelings of insecurity; rejection; deprivation of parental affections; rearing in an environment where parents constantly bicker; where one of the parents has deserted the family; where cruel or unreasonable punishment is meted out as a disciplinary measure; where sibling discrimination and rivalry exists; where parental denial of normal self-expression is present; where feelings of inadequacy exist in the sphere of scholastic or athletic attainments, or in group association.

I have further determined from my observation of adults accused of crimes and of youthful offenders examined within other settings (which also include imprisoned veterans of the armed services, those seen in hospital prison wards, the County Court, the Court of Special Sessions), that although neither psychotic, mentally subnormal nor psychoneurotic, the great bulk of these sundry violators typifies a class of individuals whose delinquent behaviour reflects an effort to adjust to varied frustrations; and it is by this manoeuvre that their cultural conflicts become translated into criminal acts. In actuality, the latter deeds represent an escape from a defeating situation and are indicative of the individual's attempt to correct a threat to his sense of self-importance. It therefore becomes the psychiatrist's province to determine the causes of the psychological pressures which find their outlet in overt acts of delinquent behaviour.

As a system restricting the individual, the social order, in its mode of dealing with the offender, should primarily evaluate his antecedent conflicting cultural development and have the latter weighed against the presenting delinquency. If the criminality is predicable upon emotional maladjustment, the home and all the other collateral environmental factors should receive clinical and social analysis (accomplishable with integrative and the indispensable aid of probational personnel) in order to determine the significance and extent of the psychophysical pressure from which the delinquent has sought relief through his antisocial or unconventional behaviour as his way of satisfying the insecurity of his own self-esteem.

Maladjusted reactors to varied and unusual stresses exist in every age group ranging between infancy and late life. Where the stressful psychosocial encounter is particularly severe, the condition requires timely and appropriate psychotherapy in order to preclude progression of the reaction to that of an established neurosis. It is this class of offenders which most frequently come into conflict with the law, and it is their psychiatric problems and difficulties which require the combined assistance of judicial, psychiatric and probational endeavours.

Under the most recent revision of American psychiatric nomenclature, currently standardized in 1952, psychological maladjustments (where the circumstantial responses are usually of temporary existence, predicated upon imminent situations and not as a rule involving basic character disorders) are diagnostically designated under the general title of *Transient Situational Personality Disorders.* These reactions are the individual's way of trying to adapt himself to an otherwise intolerable condition, and it is only when the latter remains unremitting and becomes incapable of adjustment, that a more basic disorder suggests itself.

These personality disorders, reaction patterns limited to transient situations of both serious and/or sudden impact, are expressive of violent efforts of the individual to adjust to some crushing fear or imminent rout. No neurotic or psychotic antecedent history usually exists in these individuals, and subsidence of the reactions usually takes place when and as it becomes possible for the sufferer to eliminate the impact of the trying and difficult, newly-experienced stressful situation.

To avoid unnecessary descriptive complication and because of their temporal quality and tractability to therapeutic efforts and social interposition, reference to Fig. 1 is made regarding the component reaction types of this transient disorder, the specificity of each of which is made obvious by its assigned terminology.

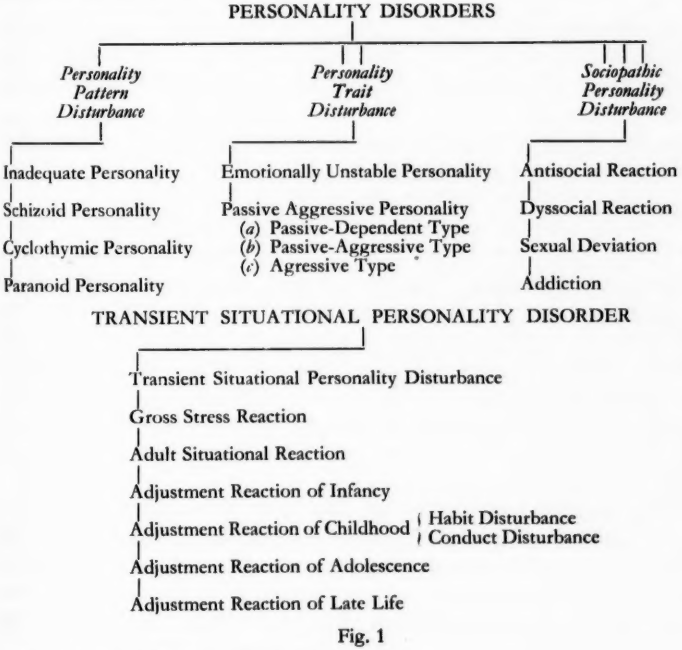

Fig. 1

But the gravity and impact of the personality defects upon our society and the enduring nature of the more malignant disorders demand detailed attention and their constituent consideration follows:

In turning our attention to the more lasting pattern and aggravated form of human maladaptation we shall begin the description by considering the personality peculiarities under the general heading of *Personality Disorders.* Under this overall designation we occasionally find offenders whose misconduct within society is the resultant of residual defects of intellectuality and emotionality due to actual brain pathology. The latter disorder can occur as a result of injury to the head, may follow brain infection, or may be due to organic disturbances in brain growth. As to head injuries, these may include concussions, contusions and lacerations of brain substance, with or without tearing of the brain envelope, and fractures of the skull with pressure of its fragments upon vital brain areas. Brain infection may include syphilis of the central nervous system, meningitis, encephalitis (sleeping sickness) and in not too frequent situations may even follow an attack of the eruptive fevers such as scarlet, measles, and similar diseases. As to disturbances in brain growth, we recognize organic brain degeneration and its pathology due to glandular disorders of the pituitary, adrenal and thyroid function. Any of these conditions can produce marked deviation in the personality and pattern of behaviour of the individual thus afflicted.

In addition to, and exclusive of, the existence of chronic brain disease, as just described, behavioural disorders, for the purpose of descriptive diagnosis, merit consideration under specific subheads in terms which will most pointedly indicate and describe the type of disturbance in each subhead.

The first of these is the *Personality Pattern Disturbance.* While the activity of the individuals hereunder may be improved by psychiatric treatment, their fundamental make-up can seldom be renovated.

Within this subhead we recognize the component 'inadequate personality'. This behavioural deviate is an individual who presents such pattern disturbances as poverty of judgment, emotional and physical exhaustion, and unfitness in both the industrial and social scope of activity. The other components of disordered personality allocable within this subhead are the 'schizoid' with their introvertive and eccentric traits; the 'cyclothymic' with their alternating moods of euphoria and dejection; and the 'paranoids' who are touchy and who impute their motives to others. Detailed expositions of the last 3 named personality types may be obtained from treatises on mental disease.

Another inclusion under the overall designation is the subhead *Personality Trait Disturbance.* Within this class of individuals we find persons with problems of emotional imbalance brought on under any condition of strain. Some of these people may go to pieces emotionally because of deep-rooted pattern fixations which preclude their effectiveness under stressful situations;

others of this class respond defectively because of a return to childish behaviour upon encountering difficulties. These emotionally maldeveloped persons are unable to accept the facts of life and to adjust themselves to them. They are unstable, irritate uncontrollably easily, and are simply babies grown up. Physically they may be fully developed, their neurotic features as anxieties or fears, if any, are not conspicuous, but emotionally they try to carry on with an attitude toward life's reality that is markedly childlike and in all respects insufficient for the demands put upon an adult responsible for his part in the world of objective existence.

Within this subhead we have the component 'emotionally unstable personality'. This behaviourally disordered unadjusted individual lacks stability under stress. In his dealings with others within his environment, in his reaction to his own culpable acts, to his own antagonism towards others, and toward his own uneasiness, he demonstrates the problem of an unpredictable emotional disposition and defective control.

Another component of maldeveloped personality within this subhead is the 'passive aggressive personality'. Such an individual's mental responsiveness to external influences may be of varying type, and quality, and exhibit characteristic problems which bear the subjoined separate descriptions:

(a) In what we term the *passive-dependent type,* we see a defenceless, irresolute individual who has a childlike readiness to attach himself to others for his own advantage and safety.

(b) In what we call the *passive-aggressive type,* we see an individual who passively indulges in inhibitory and dilatory performances, and shows an underlying reverse trend by such delaying actions as obstinacy, sullenness and inefficiency generally.

(c) In what we term the *aggressive type,* we see an individual who steadfastly continues to react to disappointment by indulging in bursts of caprice, morbid excitability and in general behaves with abnormal maliciousness. Under all this can usually be recognized a profound need to cling.

We now finally turn our attention to the most pernicious of the *Personality Disorders,* which shall be considered under the subhead *Sociopathic Personality Disturbance.* Individuals who fall into this category may be able to engage in sensible-sounding and coherent conversation, and may be able to pass satisfactory intelligence tests, but are unable to respond to the normal requirements of daily living among fellow-men. They have peculiar deep-seated conduct disorders which they express in various reactions.

The behaviour of these individuals has formerly classified them under the well-worn designation of psychopathic personality and/or constitutional psychopath. These people may be said to represent borderline psychotics or neurotics who do not justify a diagnosis of either, and operate on a precarious level of adjustment. They show no anxiety; no feeling of remorse for their acts; no discretion; impulsiveness of behaviour; sexual maladjust-

ment which is directed only towards momentary gratification; a ready ingratiation; a conditioning reflex which is difficult to desensitize, with resultant repetition of delinquent acts without learning through their previous experiences; aggressiveness against society with demonstrable desire to indulge in destructive effects by their antisocial activity; no regard for the consequences of their behaviour.

This *Sociopathic Personality* type may be descriptively differentiated under several reaction components:

The first of these is the 'antisocial reaction' type. Individuals classifiable under this component are always in difficulty; imprisonment or other punishment serves them as no benefit or deterrent; their aggressions are generally directed to authority; they are emotionally undeveloped; maintain no social relationships or devotion to any cause or purpose; are irresponsible; seek only pleasures; are insensible to the plight of others; invent explanations for their deeds and to justify their acts. When legally before the Bar charged with crime they are most usually heard to plead, and actually believe, that they have been victims of unfair treatment, have always been misunderstood, never had a fair chance, and ask for a fresh start. But sooner or later, mostly sooner, following on a fresh aggressive outburst, I have seen these personalities return to court repeatedly because of a newly perpetrated criminal act, and proffering the same kind of plea each time they are apprehended.

Another component hereunder is the 'dyssocial reaction' type. Individuals of this type, as a result of life-long existence in abnormal moral surroundings and influences, have a total disregard for society's aesthetic and moral codes. However, they are capable of faithful adherence to their own groups which comprise gangsters, vagabonds, prostitutes, racketeers and their ilk.

A further component hereunder is the 'sexual deviation' type. This particular run of deviates includes such perpetrators and personalities as homosexuals, transvestists, fetishists, paedophiliacs and sexual sadists.

'Drug Addiction' as a separate entity within this subhead requires appropriate discussion because it typifies sundry peculiarities representing a symptom picture of emotional maladjustment. The majority of drug addicts that I have observed in the various criminal courts, hospitals, veterans installations and other settings, have demonstrated disorganized individuals who take narcotics because of some personal situation which they have been unable to overcome, have tried to escape difficult circumstances and, while narcotized, acquire a questionable feeling of well-being. Their maladjustments usually begin in many ways. A small number of addicts acquire the habit by continuing the use of drugs after the need for an originally medically-administered narcotic to relieve pain had long passed. Some take drugs to repress homosexual tendencies and thereby attempt to relieve the tensions created by their conflicts. Some cannot cope with repulses incident

to amorous or marital ruin. Others acquire the habit of taking narcotics from association with other drug addicts. I have been struck by the percentage of addicts whose narcotic histories reveal that they usually begin to take drugs merely for the thrill of the adventure. I have also come to note that the addict is often an offender on other counts and that his problem, though essentially a medical one, becomes markedly complicated by the penal nature of the law he has transgressed. Not only is the drug addict a criminal because of illegal use of the drug, but because his conduct includes serious deterioration for himself and is also morally repugnant to Society. Moreover, I have often seen those addicted to narcotics brought before the court because of crimes committed in order to satisfy their craving for the drug and to secure money to buy it. As heroin is many times more potent and stimulating than morphine, I have found that the heroin addict is more prone to criminality. Finally, it has become particularly evident to me that a close relationship exists between addiction and crime, when one ponders facts that most new addicts receive their launching in the underworld.

The divergent aspects of human individuality and dissimilarity of motivation points up the continuing and increasing need for personality research in the exploration of man's forgotten, deeply buried experiences and unconscious impulses. Any type of mental illness or emotional unheaval, however trivial, affects the behaviour of the individual even though it may not result in actual criminality. Offenders are people, frequently misguided but just as frequently entangled in a sequence of situations and problems which have brought about behaviour Society frowns upon and for which it inflicts penalty as retribution.

Delinquent and/or criminal behaviour is manifestly closely bound up with the personality of the offender. The personality characteristics of the offender's make-up generally appears to instil in him a rebellion against socially accepted rules of conduct. It may therefore be postulated that his manner of thinking appears to be related to the intellectual setting within which it develops.

Psychiatric studies and analyses of persons who are unhappy, frustrated, harassed by fears and inhibitions, and even those who present disorders of perceptions (as illusions) and disorders of thought content (as delusions and hallucinations) have served to convince that human behaviour is demarcated by its drives and motivations.

One who commits a larceny of something for which he has no actual need, may be thereby retaliating at the source of ownership of the thing taken, or is by his deed carrying out a vengeful act toward sibling rivalry or toward a parent or one *in loco parentis* for whom he harbours contempt. From my investigations of those charged with crimes I have noted that many offenders (both youths and adults) whose crimes are associated with neurotic elements usually return to the place where the crime was com-

mitted. This is fairly true of kleptomaniacs, and may be said to be even more so of those engaged in such felonious undertakings as arson and homicide—particularly where sex complicated. These individuals are dependent, possess marked resentment and vengefulness toward society, together with a mixed feeling of guilt reaction for which, in reality, they seek punishment. Even the burglar who follows his craft as an occupation, may possess a deep-rooted sense of early deprivation for which he is unawarely attempting to compensate. From my observations of these perpetrators, both before and after judicial sentence, I have come to note that there exist within them these hidden inferiority feelings for which robbery (an offence requiring open aggression) or larceny and swindling are committed. This is an unconscious craving for the attainment of self-esteem through procurement of things of value for their sheer acquisition, whether they have use for them or not.

The true measure of our social greatness lies in our feelings and actions in respect of the plight of the unfortunates who are in trouble and in distress within our midst. Our people, particularly our young people, merit conservation of their best qualities, since our national character can but echo their composite expression. In this programme of conservation, and in its function for the prevention of crime, the court stands as a bulwark of understanding and humane guidance.

In the psychological study of the human race, the concept of individual differences cannot be overestimated. Each individual requires to be dealt with as a personality unique unto himself; and we must recognize that neither his problems nor his presence among us resulted from his arrival by a common road.

In its functions, the court should therefore be provided with and should have available the material tools with which to do the job. There should be a service within the court setting, of a psychiatric diagnostic screening clinic which, through examination of the accused individuals, will apprise the Bench, not only of offenders' negative elements, but will also ascertain for the court the positive side of the transgressors' character structure, as the latter may be served toward rehabilitation. It is through this forensic interaction within a functioning framework of co-operative and inter-dependent mutual relationship that the court may receive dependable diagnostic psychiatric guidance before dealing with an individual accused of crime, and receive a suggested base from which a probation programme may operate most advantageously or feasibly, for the individual as well as for the community welfare.

In the evolution of the well-being of Society, the social institution of the system of probation comprehends the method of allowing the convicted offender to go about at large where it is considered judicially desirable, on the basis of the individual's character, antecedents, age, condition of physical

and mental health, and other extenuating circumstances. This conditional release of the accused is a suspended sentence imposed by the court, aimed at encouraging and securing the good conduct and rehabilitation of the offender, qualified upon his observance of specified conditions laid down by the court at time of sentence. These terms may include judicial prescription of the accused's future environmental associations, his procurement and continuance of honest employment in approved occupation, the reporting in person at stated intervals and for a specific term to a probation officer of the court, or reporting to the court directly as and when the court may deem proper. Failure to comply with or to observe the conditions imposed, or any of them, can result in apprehension and return of the offender to court, for sentence for the original offence.

The question of age, the type of offence, as well as the offender's family and/or status, play an important part in the judicial granting of probation. It is this placing of eligible offenders under the disciplinary supervision of a probation officer, that serves both the community and the delinquent, and as a practical social plan, saves the needless overloading of jails and at the same time endeavours to rehabilitate the worthy.

The successful probation officer, while being benevolent and sympathetic in his attitude toward the probationer's shortcomings, should possess the equally important quality of being constantly aware and not unsuspecting. His basic function is not interpretation of law but interest in the social, moral and industrial adjustment of the probationer under his charge. This is truly social work and involves such rehabilitative working relationships as collaboration with local social organizations, exploration of neighbourhood recreational facilities, as well as reference of probationers to appropriate medical agencies where health problems may complicate the picture of rehabilitation. The officer should take a realistic attitude and recognize that the delinquent's behaviour is frequently the indicator of some firmly implanted psychological or social necessity which has remained unfulfilled and has now launched the offender to a level of conditional release from legal custody.

In the nature of things certain offenders' sentences, in a great measure, include the privilege of judicially granted probation. It is to be hoped that judges will uniformly come to regard that the probation officer's task is one of specialized social work. This, particularly in view of the peculiar fact that, as compared to voluntary intake procedure between social case worker and client, the intake procedure for offender probationers is involuntary. While most social welfare clients welcome help, the probationer does not ask for help but has it thrust upon him. Thus the probation official is obliged to employ every possible skill of case work in assisting the offender to help himself toward re-adjustment. When the probationer's problems also reflect deficiency or impairment of emotional content and personality maladjustments, recognition by the officer of these complicating attributes

becomes imperative as these conflict situations have added need for the techniques and insights of psychiatric study and therapy.

I note with considerable satisfaction the contents of the article which had appeared in *Mental Hygiene News* of October 1953, published by the New York State Department of Mental Hygiene. It reports the results of a general full week's session held at St. Lawrence University. The Institute was attended by some 450 workers in the fields of correction, parole, probation, mental hygiene, social welfare, youth work and allied fields.

I quote in part from that article:

'The line between criminality and mental illness or defect is being redrawn . . . as a result of advances that have been made in psychiatric and psychological techniques and the increasing use of these techniques by social agencies, probation officers and courts, especially in the New York City region. When delinquent behaviour of children brings them under study, and it is found that the behaviour has its roots in disturbances of emotional functioning or intellectual development . . . there is a growing tendency to diagnose them . . . not as delinquents but as mentally ill or defective. . . . Policemen are becoming increasingly aware that the juvenile criminal act is often not so important as the social and psychological factors behind it. . . .'

From my own observations, I not only concur in these conclusions but am persuaded to extend the findings to relate to adult offenders who, 'tis trite but true, are children who became grown up. Finally, the following utterance from a passage in the words of Dean Emeritus Roscoe Pound of Harvard Law School, remains as valid to-day as when it was enunciated many years ago:

'Despite all that the criminologists have done in the last generation the criminal law is still framed chiefly in terms of punishing the vicious will. Despite all that criminologists and physicians have shown as to the necessity of special institutions with expert management for many classes of delinquents, the legal theory of ideal equality before the law leads the criminal law, whenever it is in the hands of lawyers, to consign all to a common prison. Despite all that psychiatry and psychology have achieved, the lawyer can draw only a plain straight line . . . of . . . artificial legal conception of normal responsibility. . . .'

> 'Think on this doctrine—that reasoning beings were created for one another's sake; that to be patient is a branch of justice, and that men sin without intending it . . .
>
> A wrong-doer is often a man that has left something undone, not always he that has done something . . .'
>
> *Marcus Aurelius*

Application of Blood Grouping in the Examination of Blood Stains*

Briggs J. White, Ph.D.

Advisory Chemist, Federal Bureau of Investigation, Washington, D.C.

It has long been the experience of the serologists in the F.B.I. Laboratory at Washington, D.C. that one of the most important and most frequently used phases of medico-legal work is the scientific examination of blood-stained evidence to determine the blood classification of suspected blood stains. This experience is based on the examination of evidence secured in the investigation of criminal cases in which the F.B.I. has jurisdiction and also from law enforcement agencies from all parts of the United States and its territories.

As a result of information obtained from such analyses, murderers, rapists, hit-and-run drivers as well as other perpetrators of crimes of violence have often been successfully brought to justice by the identification of blood stains on garments, weapons, automobiles and many other types of evidence. Information obtained from such examinations is presented in the form of court testimony by our scientists at trials and courts-martial in a large number of these criminal cases. I mention this experience because many of the conclusions and procedures described in this paper are based to a large degree on control experimental tests which are made simultaneously with those which are conducted on evidence in criminal cases in addition to specific research work conducted in the field.

The testing of dried blood stains to determine the blood classification is a complex and time-consuming procedure. The difficulties involved are often not appreciated by persons who, although they may have considerable experience in testing liquid blood, have not made a complete study of the techniques necessary when the examination of dried blood is involved. Our experience has definitely indicated that attempts should not be made to classify dried blood stains unless the serologist has made exhaustive tests on similar stains. The difficulties involved in conducting such examinations are particularly acute when applied to blood stains

* Presented at the Sixth Annual Meeting of the American Academy of Forensic Sciences, Chicago, 25-27 February, 1954.

secured during the investigation of criminal cases, because no control is possible over the conditions under which such evidence is subjected. It is often necesary to make examinations of blood-stained clothing which is dirty and the stains may be contaminated by grease and other interfering materials. The presence of dyes and sizing also contributes to the difficulties involved. As a general rule the tests for isoagglutinogens are more reliable than the methods which test for isoagglutinins because of the greater stability of the former materials.

Until 1943 our Laboratory had been using the tube absorption method for grouping dried blood as a means for testing for isoagglutinogens. It was determined that this method sometimes will give rise to erroneous results, particularly when the tests were conducted upon old or contaminated blood stains. These tests would sometimes fail to identify a weak isoagglutinogen particularly in Group AB blood which was especially weak in either the A or the B factor. Wiener and Kosofsky [1] pointed out that a more accurate standardization of the sera used in grouping of stains would lead to more consistent results. The method used for standardization of group-specific substances in such sera was a titration of the sera with saliva from secretors of known groups. By this method it was possible to select sera which would yield the maximum sensitivity at a specific concentration. The tube absorption method also had the distinct disadvantage that a relatively large amount of dried blood was necessary to permit a blood grouping. For this reason it was usually not possible to group blood stains on a subject's clothing because such stains were usually rather small in size and sufficient blood was not present for analyses. Wiener [2] applied such standardized sera to the grouping of dried blood stains using much smaller amounts of blood.

This method with some modification was used experimentally in our Laboratory on 250 different specimens of blood-stained cloths representing 49 different blood sources. The samples used were stained cloths containing blood from 23 cadavers and 26 living persons, and the stains varied in age from ½ year to 3 years. As a result of these tests it was ascertained that such blood grouping tests were reliable when conducted on control cloths stained with blood from the 4 major blood groups. It was possible to classify all 250 specimens correctly, using this method. The group of the stain was not known to the serologist at the time the tests were made. It is pointed out that such controlled tests, while they indicate that careful tests can be done 100% successfully, do not represent the difficulties encountered when similar tests are made on evidence secured in criminal cases. Contaminations, which were necessarily present on much blood-stained evidence actually submitted for laboratory analyses, limited the percentage of successful classifications which could be made on such evidence. Obtaining the necessary sensitive sera after titration with saliva was a long and extremely tedious process. For this reason, other sources

were tested in order to obtain materials which would give the necessary sensitive sera.

In 1946 anti-A and anti-B isogglutinin plasma fractions were obtained from the Harvard Medical School for test purposes. When those isoagglutinin fractions were dissolved in saline solution and diluted to the proper concentration, it was determined that they had many advantages over commercial sera which could be purchased at that time and over liquid sera prepared in our Laboratory. These materials had the great advantage that they could be made up to a certain concentration in saline solution and kept over long periods of time in a refrigerator without changing titer. Tests indicated that successful classifications could be made of dried blood stains on cloth when two pieces of the stained cloth approximately ¼ inch square were used. The same results were obtained when 3 mg. of dried blood scrapings were used for the tests. Using such small amounts of blood, it is necessary to have the titer of the serum remain constant. It is now possible to use diluted concentrations of most commercial sera for these purposes because of the increased quality of such sera within recent years.

In general, the tests for isoagglutinogens in dried blood are based on the capacity of the blood stain specifically to absorb the agglutinins present in the serum added to the blood. This absorption is usually allowed to take place for about 12 hours. The tubes containing the dried blood and sera are then centrifuged and the liquid transferred to a multicell plate. The absorption or non-absorption obtained is then determined by adding one drop of a dilute solution of sensitive A and B cells to each sample. The plate is rotated at a constant speed for a previously determined period of time and the results obtained by reading the agglutination microscopically. It is obvious that the percentage of clearly defined positive results will be lower when the tests are made on blood-stained evidence which may be contaminated than on clean control cloths which are prepared in the Laboratory. In order to increase the percentage of these positive grouping tests, attempts have been made to control the various factors which arise during the examination of evidence in criminal cases. In order to arrive at any valid conclusions about which control measures may actually be of value in conducting dried blood grouping tests, it is necessary to apply such controls to a large number of specimens. Such research is usually accomplished in our Laboratory in conjunction with the daily tests which are being made on current examinations. This permits an evaluation of such control procedures on a large number of known blood samples over an extended period of time and under the varying laboratory conditions. Approximately 2,000 blood examinations are conducted in the F.B.I. Laboratory each year on evidence secured in connexion with the investigation of criminal cases. A large percentage of these examinations involves the grouping of dried blood stains.

Some control measures which have been found necessary in conducting dried blood grouping tests are described as follows:

1. Some type of titration of sera and determination of the desired dilutions in order to ascertain the maximum sensitivity point of the sera. It is desirable to obtain a grouping serum which will not change titer when diluted with saline and the serum is allowed to remain in a refrigerator for some time.

2. It is necessary to keep the concentration of blood as nearly constant as possible. This control is difficult because of the varying amount of blood in dried stains but it can be controlled by changing the size of the sample.

3. The size of the drop should be uniform in order to keep the amount of serum added to each specimen constant.

4. There should be a constant rotation of the slide to which the grouping sera and the known red blood cells are added. This is accomplished by a mechanical rotator set for a definite period of time which has been previously determined for the particular sera being used.

5. The tests should be carried out at approximately the same temperature and humidity.

6. It is necessary to use blood cells which are known to possess a high degree of agglutinability.

7. Control blood-stained cloth or dried powdered blood samples comprising the 4 major blood groups together with the A_2 and A_2B subgroups shall be run simultaneously. It is desirable to use controls which include dried bloods which are aged to a greater extent than any of the blood which might be present in the questioned samples.

8. If blood stains are present on cloth, it is necessary to run control samples of the unstained material closely adjacent to the stains in order to ascertain whether there are any substances present in the cloth other than the blood stains which would cause absorption. If clothing is dirty or visibly contaminated, even more care is necessary in running control samples around the blood-stained areas.

Using all of the above-mentioned control measures, it is usually possible to arrive at a definite conclusion concerning the blood classification of suspected blood stains. There are instances, of course, when the control pieces of cloth absorb on either, or both, anti-A and anti-B sera. The blood stains can then only be reported as unsatisfactory for the grouping procedure.

During the past few years attempts have been made to determine other factors which would cause the results of the blood grouping tests to be unsatisfactory. It has been noted that a higher percentage of false absorption has been obtained in stains on certain types of cloth, particularly khaki and similar materials. It is known that any appreciable deviation from the neutral pH will have an adverse effect on blood grouping procedures. In order to minimize the possible effects of acidic and basic substances which might be present in the cloth, buffer solutions have been used in the grouping sera solutions. Phosphate buffers with a pH of 7 are used to prepare the grouping sera so that the solutions are isotonic. Buffering of the grouping sera appears to have the effect of minimizing any variation in pH and, therefore, produces an increase in the percentage of satisfactory results. It has also been found that the addition of buffer solution to cells

instead of saline as a suspending medium, retards hemolysis of the cells and permits their satisfactory storage for a longer period of time.

Blood stains, occurring in the form of crusts, may be removed from the surface of the material and pulverized. This powdered blood can then be tested in the same manner as the small pieces of blood-stained cloth. It has been found that approximately 3 mg. of dried blood will be sufficient for testing if sensitive grouping sera are used. When the blood stains are spread over non-absorbent surfaces in a thin film so that it is not possible to obtain sufficient amounts of uncontaminated blood, certain control procedures are desirable. Wiener[3] suggests that the blood stains be dissolved by sucking a few drops of the testing serum in a capillary pipette back and forth until the stain is completely dissolved or suspended. As a control for this procedure the same amount of the testing serum is used to treat an unstained portion of the surface in order to detect any contaminating materials present in or on the material which would cause false absorption.

Summary

The value of conducting dried blood grouping determinations on evidence secured in the investigation of criminal cases has been well established.

Improvements in the various methods and techniques used have resulted in a large increase in the percentage of positive results obtained in actual examination of evidence.

It has been determined that various control procedures are necessary in order to eliminate the possibility of extraneous substances causing false absorption. These control measures include:

1. Titrating sera to ascertain the maximum sensitivity point.
2. Controlling the concentration of blood in sample.
3. Keeping the amount of serum added to each specimen constant.
4. Rotating the grouping slide constantly.
5. Controlling temperature and humidity.
6. Using blood cells with high degree of agglutinability.
7. Using control blood-stained cloth or dried powdered samples.
8. Running control samples of unstained material closely adjacent to blood stains in order to detect any false absorption.
9. Using buffer solutions in preparing grouping sera and suspending test cells in order to minimize possible effects of extraneous acidic and basic substances.

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Electroencephalography and Forensic Medicine

I. Introduction to Electroencephalography

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It is inevitable that discoveries and advances in psychology will encroach upon problems relating to the regulation of society, since one of the major aims of psychology is to understand the origins of behaviour, whatever its form. As this understanding grows, so is the psychologist forced to accept a form of determinism in behaviour. His science endeavours ultimately to predict behaviour, and prediction depends upon probabilities which can be stated precisely. As one result of this kind of study it is now being recognized that people vary in the extent to which they can control their impulses and urges, so that some are wholly capable and others wholly incapable of restraint, with a continuous variation in degree of restraint between these two extremes. Variations may also occur in the same person from time to time. The factors determining this ability of restraint arise from both environmental and heredito-constitutional sources, and the outcome is the result of a complex interaction between the two. A powerful and persisting environmental influence is provided by society in the form of rules and regulations, and one of the functions of these rules is to restrain such socially dangerous attitudes as 'I do what I do because I am what I am', which may always arise when the bogey of determinism is invoked. Despite this, the rules of society must be modified gradually in accordance with broader appreciation of the determinants of behaviour, for otherwise they will contain terms or implications which are at variance with contemporary knowledge. The memorandum given by the British Medical Association to the Royal Commission on Capital Punishment in 1950[5] is an excellent example of the operation of this process.

One of the more recent technological advances in the study of factors behind behaviour and its aberrations is the electroencephalograph, or EEG,* a machine for recording the electrical rhythms of the brain. This paper

* 'EEG' is also used to refer to the electroencephalogram, i.e. the tracings recorded by the machine.

endeavours to review current knowledge about electroencephalography in relation to crime, antisocial behaviour and law.

In the interests of clarity, the author has found it necessary to divide the paper into two parts. The first is an introduction and survey relating to the whole subject of electroencephalography, whilst the second deals with its general and specific forensic implications: a full appreciation of the latter is impossible without some such reference framework as is supplied by Part I.

Before proceeding with the main substance of the communication, it is perhaps well to recall the conclusions of the United Nations Seminar in Brussels, 1951,[9] which dealt with the implications of medico-psychological and social examination of delinquents. By examination is meant the application of scientific method by a person or persons having expert training and experience in the use of that method. The general aim of the examination is to provide the judge with data that will help him:

i. To determine responsibility in so far as the methods used can shed light on this legal issue;

ii. To give full weight to both obvious and unconscious motivations behind the crime;

iii. To determine the most appropriate sentence, and to appreciate the medical, social and psychological issues affecting all therapeutic measures influenced by his decision.

Concerning the EEG, it was concluded (p. 88):

'This provides a new method of recognizing effects of old injuries or diseases of the brain or the presence of epilepsy. Positive findings are more important than negative ones. *It is an adjunct to, and not a substitute for other methods of examination.*' (Italics inserted.)

I: Electroencephalography

History

In 1929, Hans Berger[3] reported that it was possible to record an electrical rhythm from the human head, and that this rhythm originated in the brain. He used a string galvanometer * in his early studies, and checked the validity of his tracings by comparison with those obtained through trephine openings in the skull. Despite the extreme care and scientific caution with which Berger undertook his work, his reports were treated with considerable scepticism until confirmed by Adrian and Matthews[1] in 1934; it was then generally accepted that the brain of man has an electrical beat arising from neurones and appearing as a mixture of more or less sinusoidal fluctuations ranging from 1–60 cycles per second (c./sec.), being clearest at about 10 c./sec. in normal adults. Berger called this 10 c./sec. rhythm the

* For a glossary of terms which may be unfamiliar, see Appendix I.

'alpha' rhythm, and activity at 15–60 c./sec. 'beta' rhythm. He showed that the alpha rhythm changed with age, sensory stimulation and with various physico-chemical changes in the body. In particular, he noted that the alpha rhythm was greatest with the eyes closed and tended to disappear when they were opened. Berger used two electrodes for recording his EEGs, one at the forehead and one at the back, giving the erroneous impression that the rhythms came from all over the head. Subsequent observers have demonstrated that there are in fact several different rhythms arising from various parts of the brain. The basic method for doing this was first used by Adrian and Matthews,[1] who (by employing a number of fairly closely-spaced electrodes) were able to show that the origin of the alpha rhythm is near the visual association areas of the occipital lobes of the brain. Details of these location methods are presented below.

Recording an EEG involves affixing electrodes to the head and connecting them to an amplifying system, which is in turn connected with a recording system. Ideally, the recorders faithfully reproduce signals from the brain which have passed through the scalp and have been picked up by the electrodes, precautions being taken to ensure that as few extracerebral changes as possible are recorded (see Glossary under Artifact). Unfortunately, as is discussed in the section dealing with methodology, there are many problems and difficulties in achieving these simply-stated goals.

The electrical activity of the brain, as any other type of physiological activity, is a manifestation of physico-chemical disturbances in cells and in cells organized into tissues. Electrical changes are accompanied by chemical, thermal and mechanical changes, but the electrical are the easiest to measure, since they readily pass through tissue barriers and may thus be rendered apparent at a distance; furthermore, electronic techniques are far ahead of any others in dealing with minute changes. Electroencephalography is thus a branch of electrophysiology, which involves the recording of electrical disturbances generated by living tissue, and the stimulation of living matter by electric currents.

Since 1934 a vast amount of work has been done on the application of electroencephalography, relating in particular to neuropsychiatry. Here its value has been immense, especially in the fields of epilepsy and allied disorders, and in the localization of lesions in the brain; for in both these categories the electrical activity of the brain may show characteristic disturbances which are of considerable value in ultimate diagnosis and treatment. The data of this work have resulted in a fairly systematic set of definitions and classifications of the various rhythms and complexes encountered in electroencephalography and the salient details of their collection are obtainable in a number of works.[7, 13, 14, 19, 20, 25, 31] Here it will be sufficient to give a brief outline of the conclusions and definitions generally accepted in 1954. We will begin with the normal rhythms of the brain.

The Alpha Rhythm. This is defined as electrical activity between 8 and

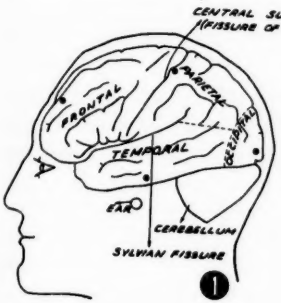


Fig. 1: Topography of the left hemisphere of the cerebral cortex. All that part of the hemisphere in front of the central sulcus, and above the Sylvian fissure is the *frontal lobe.* The *temporal lobe* lies below the Sylvian fissure, its other boundaries being here delineated by broken lines. The most posterior portion of the cortex forms the *occipital lobe,* its forward boundary also being indicated by a broken line. The remaining part, lying between the frontal and occipital lobes and above the temporal lobe, is the *parietal lobe.* The four hatched circles represent the standard electrode positions used in the author's laboratory, viz. frontal, parietal, occipital and temporal.

13 c./sec. inclusive arising from occipital or parieto-occipital areas,* normally being diminished ('blocked') during visual stimulation and often during other types of sensory stimulation, mental effort and attention. Amplitudes of alpha rhythm range from 3–100 microvolts (μv), with an average of about 30 μv, whilst the duration of its presence, known as the alpha index and expressed as 'per cent. time alpha' varies from 0–100 in different persons, with an average value of around 55. The most constant feature of the alpha rhythm is its frequency, which rarely varies by more than 2 c./sec., usually less than 1 c./sec. over periods as long as several years. Comparable constancy has been reported concerning alpha amplitude and per cent. time, although with these two variables there may often be considerable fluctuation within short periods, since they are particularly susceptible to sensory stimulation and other normal environmental influences. As a general conclusion, however, it may be stated that the alpha characteristics of an individual under experimental conditions show a high degree of diurnal consistency such that one individual can be distinguished from another by his brain potentials. Although EEG individuality is not as complete as that of a fingerprint, studies of twins have revealed certain features common to both members of an identical twin pair, these being noticeable in the chief characteristics (frequency, per cent. time, amplitude and responsiveness to stimulation) of the alpha rhythm. This has led to the conclusion that the EEG reveals patterns of cerebral organization which are strongly influenced by hereditary factors.[22, 23]

With the development of more refined and precise recording and analytic techniques it has now been established that the alpha rhythm is rarely monorhythmic: 2, 3 or more separate components can usually be detected, often with different spatial locations and orentiation within the head; furthermore, these alpha components may show individual or separate responses during various kinds of sensory stimulation or mental activation.

It must be noted that rhythms of alpha frequency have often been reported as arising in frontal, central, parietal and temporal areas. These

* Fig. 1 illustrates the regional topography of the surface of the brain.

should not be referred to as alpha rhythms, since their meaning in terms of cerebral function is probably quite different. They are discussed in more detail below.

Beta Rhythm. In comparison with the alpha rhythm, far less is known about the low voltage fast rhythms originally named beta waves by Berger. A particular problem relating to any fast activity in the EEG is that the frequency of muscle potentials is of a similar order, namely from 14–50+ c./sec., so that sometimes it is extremely difficult or impossible to distinguish between cerebral and muscle activity. This has often given rise to controversy about reports dealing with fast activity and it is very probable that some of the early EEG studies did confuse muscle activity with cerebral rhythms.

At present, beta rhythm is broadly defined as activity between 14–30 c./sec. arising from any area of the brain. Its amplitude normally ranges from 1–20 μv. It has generally been reported as more prominent over the precentral and frontal regions than elsewhere, although a study from our own laboratory [26] revealed no strict confinement to anterior regions among normal adults. This study also revealed a mean beta frequency of 18 c./sec., and a mean amplitude of 9 μv. It is now clear that there may be several kinds of beta rhythm in the EEG, and that these have different functions or meaning according to their location and responsiveness to stimuli. In particular, it should be noted that Jasper [21] has reported marked beta rhythms from the motor regions of the exposed human brain during operation, and that these rhythms showed a blocking response (similar to that of the alpha rhythm) during changes in limb position and at the initiation or cessation of prolonged limb movement; comparable changes have been observed through the intact scalp. Finally, several classes of beta variation have been reported during a wide variety of conscious activities, such as eye opening, mental imagery and mental arithmetic,[26] whilst there have been several reports of increased fast activity during hyperemotive and anxiety states.[19]

Theta Rhythm. Before 1944, rhythms with a frequency of 7 c./sec. or below were called 'delta' rhythm or 'slow alpha' rhythm, but in that year Walter and Dovey [33] proposed the term theta rhythm to designate activity at 4–7 c./sec., found in records from patients with subcortical tumours and which is apparently often peculiarly related to disturbances in cortico-basal circuits. The term was later extended to include rhythms in the same frequency band which are associated with functional rather than organic disturbances, whilst more recently there has been a tendency to describe almost all rhythms at 4–7 c./sec. as theta rhythm.

Theta rhythm is a dominant feature of the EEGs of children in the 2–5 year age group and is normal in children up to the age of 16 years. Traces of theta rhythm can be found in about 65% of young normal adults, but there is a decrease of normal theta rhythm with old age.[26] Theta

rhythm may arise from any area of the head, particularly the parieto-temporal areas. It may show variations during sensory and emotional stimulation, also during mental activity, particularly when this involves emotional changes. A great deal of evidence suggests that theta rhythm is intimately related with cortical maturation processes, which would account for its gradual decrease with increasing age.[16, 18, 19]

Delta Rhythm. This term was originated by Walter in 1937 [30] to designate high voltage slow waves at 0.5–3.5 c./sec., found in the region of brain tumours. There is now a tendency for it to be used to describe all rhythms in this frequency band. Delta rhythm as a normal constituent of the EEG is found in infants up to the age of 2–3 years and during sleep in all age groups; its voltage may range from 10–250 μv or more, whilst often the waveform is irregular due to the presence of several components. Abnormal delta rhythms are common in a variety of organic cerebral disorders, epilepsy and several other pathological states.

Other Spontaneous Rhythms and Activities. There are numerous combinations of rhythms or activities which form distinctive EEG patterns and are often associated with some kind of neuropsychiatric disturbance. These have been discussed in detail by the author elsewhere,[27] together with an appraisal of the clinical significance of electroencephalography and with illustrative examples of the various abnormalities, so that brief allusions will be adequate here.

The characteristic abnormalities in epilepsy are the 3 per second wave-and-spike, often associated with *petit mal*; the slow spike-and-wave (0.5 to 2 per second), usually associated with organic disturbance and always with a history of some kind of seizures; spikes or sharp waves, which are often focal and associated with acquired epileptic disorders related to cortical irritation; paroxysmal disturbances, either slow (less than 7 c./sec.), fast (more than 14 c./sec.) or of mixed frequencies, the latter giving rise to a 'dysrhythmic' appearance, all three being common in a variety of epileptic conditions. Significant abnormalities encountered in organic cerebral diseases are focal slow disturbances, which may be divided into delta and theta. The former is the characteristic activity surrounding a tumour focus in the cortex of the brain. The latter is encountered in cases of deep, subcortical lesions. There are many other significant abnormalities encountered in the EEGs of clinical material, and the reader is referred to the available text-books [14, 19] for a fuller appreciation of their nature and clinical significance. Those abnormalities which are of specific forensic significance will be discussed in detail in Part II of this paper.

Of the many other rhythms which have been reported, it is perhaps relevant to mention a few which may possibly (though by no means certainly) be associated with psychological instabilities, disturbances or deficiencies.

Firstly, there are the 'alphoid' (8–13 c./sec.) rhythms which are some-

times present in the frontal areas. It has been claimed that these are associated with emotional instability and immaturity,[24] whilst both Gallais *et al.*[10] and Mundy-Castle *et al.*[28] have reported relatively high incidences of such activity in uneducated, non-Westernized normal African adults. Also, Darrow[6] has claimed that there is a tendency towards an increased anterior dominance of alpha-like (i.e. alphoid) activity in emotionally less stable individuals.

Other rhythms which appear to be associated with certain psychological disturbances or disorders are the *rhythme en arceau*[12] and the alpha variants.[15] The former is confined to the rolandic region of the brain, possessing a frequency of 9 ± 2 c./sec. and an amplitude of 20–40 μv. It has a characteristic hairpin-like form due to the fact that each positive phase is accentuated and sharp-tipped, whilst the negative phase is rounded, hence its descriptive name. The *rhythme en arceau* is rare in normal persons, but occurs in a variety of clinical conditions, and is usually associated with one or more of the following psychological characteristics: hyper-emotionality and emotional lability; hypermotility; anxiety; neurosympathetic disturbances.

There are two types of alpha variant, a fast and a slow, each being harmonically related to the alpha rhythm with which it is associated. Thus a slow alpha variant of a 10 c./sec. alpha rhythm would possesss a frequency of 5 c./sec., a fast alpha variant one of 20 c./sec. Alpha variants have been reported in association with a wide variety of symptoms, such as headache, emotional and vasomotor instability, disturbances of consciousness and personality, as well as epilepsy. Hill[17] has classified the slow alpha variant as a maturational defect, reporting an incidence of approximately 1% in a normal population.

Berger was the first to report that the EEGs of children are basically different from those of adults, in that they contain very much slower frequencies, and many early studies indicated that in general, the younger the child the larger and slower are the electrical rhythms of its brain. Walter[19] applied automatic frequency analysis to a group of 110 normal children, and was able to demonstrate that although there is an apparent gradual increase in frequency of rhythms with age, this is not due simply to a progressive acceleration but to a variation in prominence of a number of co-existing rhythms of slow and faster frequencies. This had not been fully appreciated until the development of the Walter automatic analyser was complete.[2] This apparatus enables a rapid breakdown of the EEG pattern into its component rhythms and their different frequencies, locations and changes with stimulation or state. The following are the main conclusions derived from the application of this technique:

1. Delta, theta and alpha frequencies are present in the youngest age-group.
2. Diffuse delta rhythm is dominant until one year and persists as a subdominant component up to 5–6 years.

3. Parieto-temporal theta rhythm is dominant in the 2–5 year age-group, thereafter being very small or intermittent. Theta rhythm is usually augmented by closing the eyes and by emotion (e.g. laughing, crying, etc.) in children up to 2–3 years.

4. Alpha rhythm is present in infancy. By the age of 5–6 years it is approximately equal in dominance to theta rhythm. Its frequency is 8–9 c./sec. in the lower age-groups, 9–10 c./sec. in the older groups (8–15 years). It is rarely responsive to visual stimuli below 3 years of age. It is more widespread in its area of distribution in younger children than older ones, and in general it is monorhythmic.

A striking feature of the EEGs of young children is the predominance of theta rhythm, particularly in its variation with affective changes. Walter [19] has claimed that in those children in whom there is a clear connexion between affective changes and theta rhythm, visceral and other autonomic activity may play an important part in determining the amplitude and regularity of the rhythm. He comments on the emotional as opposed to intellectual determination of behaviour in young children, and puts forward the hypothesis that theta rhythm may reflect the level of maturity of the mechanisms linking the cortex, thalamus and hypothalamus. This hypothesis incorporates the observations of Hill [16] that theta rhythm from the temporal areas is common in the EEGs of aggressive psychopaths, this leading Hill to suggest that adult aggressive psychopathy may be produced by a failure of development in the central nervous system. Thus, Hill and Watterson [18] pointed out that many of the records of young psychopaths would pass for normal in children; furthermore, one of the commonest features of the EEGs of problem children is an excess of theta rhythm, the same kind of abnormality as found in the adult psychopath, yet all children with such disorders do not become psychopathic adults. This is further evidence in favour of a cortical maturation process.

The 10–10.5 c./sec. adult mean alpha frequency norm is reached by about 13 years, whilst the general features of the adult EEG are fairly well established by 17 years. As has already been mentioned, normal theta rhythm shows a continuous decrease with advancing age, this being attributed to a continuing maturation process. Little is known about the development of beta rhythm in the EEG. There is no significant difference between its incidence in adult youth and normal senility.[26] The alpha rhythm shows several changes with old age, notably a significant decrease in per cent. time amplitude and frequency, the latter dropping to a mean of 9.39 c./sec. by the age of 75 years.[29]

Methodology

Technical Aspects of Recording

There are certain basic principles underlying the accurate measurement of bio-electric phenomena, the most fundamental being electrochemical. The

body is in effect an electrolyte, i.e. a compound susceptible to decomposition by an electric current. Whenever a chemical reaction takes place within the body, or energy is transformed or transferred, electrical changes also occur. These electrical changes can be picked up by placing electrodes in contact with the electrolyte (body), but if the metal of a pair of such electrodes is not exactly similar, it is possible for a current to flow between them. Furthermore, if a potential difference occurs between the two electrodes, this may cause an accumulation of ions round the positive and negative electrodes, which will tend to neutralize the potential difference—a phenomenon known as polarization. Since these factors will distort any picture of the bio-electric activity picked up by electrodes on the body, it is necessary to reduce their influence to an absolute minimum. This can be done by using non-polarizable electrodes, the most satisfactory being made of silver coated with silver chloride.

The next considerations are the amplification and recording of the signals picked up by the electrodes. Certain standards for EEG equipment have been drawn up by both the British [19] and American [8] EEG societies, and it is most important not only that equipment should accord with these specifications, but that there be full understanding both of their limitations and the effects on signals of changes in the various settings incorporated in the machine. The majority of EEGs in use today possess 6, 8 or more amplifying channels, thereby enabling simultaneous recording from many different parts of the head.

With regard to electrode placement and spatial location of the origins of rhythms from within the head, there are two forms of electrode disposition, known as 'bipolar' and 'unipolar'. With the former, each recorder is connected to a pair of electrodes, both of which are placed over active brain, whilst with the latter one of the pair is over active brain and the other, called a 'reference' electrode, is situated at a relatively inactive point. There has been considerable controversy about the merits and demerits of these two methods, with the most penetrating criticisms aimed at the unipolar system. The contemporary view is, however, that in certain circumstances the use of a reference electrode is justified provided that:

(a) Initial location of the focus is by the bipolar method;

(b) The least misleading reference electrode is used (e.g. 6th or 7th cervical vertebra); and

(c) The limitations of the unipolar system are recognized.

Location by the bipolar method is effected by connecting electrodes in a chain, so that an electrode common to two of the EEG channels has both a positive and a negative input lead attached to it. This means that if this common electrode is immediately over a source of electrical potential changes, then the activity recorded by the two channels will be of equal voltage but *out of phase*, i.e. the two tracings will be mirror-images of each

other. The point beneath this common electrode is known as a focus, and the technique for locating it in this way is known as the phase-reversal method. It is important to note that the voltages from a focus need not be greatest from those channels closest to it, since recorded amplitudes are a function of several additional factors other than proximity to the signal generator.

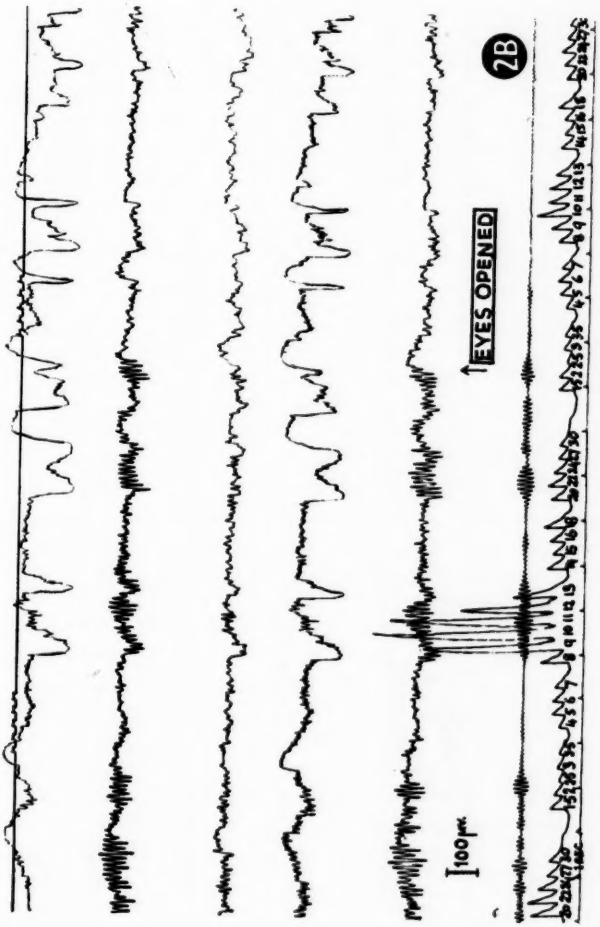
Waveform Analysis

Since EEG records are almost invariably complex and possess a number of contributing components, accurate analysis presents several problems. The classical method of analysis in electroencephalography has been visual, with the aid of a transparent ruler or cursor. Its chief disadvantage is that the limit of visual resolution of a complex waveform does not permit identification of all components which may be contributing to it: e.g. low voltage rhythms may be masked by larger components, and if several rhythms of different frequencies are present, an infinite variety of waveforms is possible. Whilst some of these difficulties can be resolved by careful electrode placement (thereby separating out the different components according to their origin) or by changing the physiological or mental state so that individual rhythms are differently affected (e.g. opening the eyes will block the alpha rhythm yet may leave theta rhythms unchanged), the most satisfactory solution is to incorporate some form of automatic frequency analysis. This can then be used effectively in conjunction with the aforementioned aids to visual analysis.

The most satisfactory frequency analyser for EEG work is that developed by Walter,[2, 19] now used in a number of EEG centres. In Fig. 2 are shown 3 sample EEG strips, 2 of which possess superimposed frequency analysis write-outs. To give the reader a general idea of what a normal EEG looks like, these samples are respectively from a normal child, a normal adult woman and a normal senile. Short descriptions of the samples are incorporated in the legends. The following explanation will assist in interpretation of the output from the frequency analyser, the write-out of which is situated at the foot of the EEG above that of the time marker. The analyser trace gives a spectral display of the frequency analyses broken up into 10-second epochs, the channel analysed being indicated by the number of deflections in the topmost line, e.g. 3 deflections = channel 3. There are 24 upward deflections in each 10-second epoch of the analyser write-out, each deflection relating to a tuned circuit associated with a storage register: the relative amplitudes of the 24 deflections thus reflect a frequency spectrum derived over a 10-second period, a minimal upward deflection occurring if none of a particular frequency is present. The actual frequencies to which these upward deflections correspond are indicated in Fig. 2. A long downward deflection is provided at the beginning and end of each analysis epoch, whilst shorter downward signals between 3.5 and 4 c./sec., 7 and 8 c./sec.,

Fig. 2: EEGs of Normal Subjects. (Note: Unless otherwise indicated, all samples are recorded from electrode derivations as shown in Fig. 2*c*.)

2*a*. Child aged 3 years. Dominant rhythms are at 5–6 and 8–9 c./sec., amplitudes ranging between 60–120 *μv*. Most of the slower activity, recorded at 2–2.5 c./sec. by the analyser, is due to movement artifact involving both head and eyes: voltages generated by movements of the latter are seen most clearly in channels 1 and 4, i.e. right and left fronto-parietal (the channels are numbered from above downwards); some of the very fast activity superimposed on these slow waves is artifact from muscles in the scalp and round the eyes. Note that eye opening causes diminution in both theta and alpha rhythms, this being reflected most clearly in the analyser trace.



2b. Adult female aged 28 years. Classical alpha rhythm at 9–11 c./sec., average amplitude around 80 *μv.* Clear blocking of alpha rhythm on eye opening. Channel 6 is connected to the resonating circuit of the frequency analyser, and oscillates only when that frequency occurs in the channel being analysed (5). Note the increased eye movement artifact on channels 1 and 4 whilst the eyes are open and therefore blinking or roving. Traces of low voltage muscle artifact are also apparent, being fast and spikelike.

2c. Normal male aged 75 years. Alpha rhythm at 8–9 c./sec., amplitude around 15–20 μv (amplification in this record is much higher, as shown by the 100 μv calibration mark): such reduction in alpha amplitude is a natural feature of old age. Clear blocking of alpha rhythm with eye opening. Eye, muscle and movement artifacts are easily distinguished, the last two occurring as the subject coughed towards the end of the sample.

13 and 14 c./sec., 18 and 20 c./sec., indicate the various frequency bands, i.e. delta, theta, alpha, low and high beta.

Facilities are also provided in the machine for recording activity which is out of phase *between* two channels of the EEG, and for obtaining an average analysis over periods of up to two minutes. It is also possible to observe the output of any one of the resonating circuits thereby enabling a selective recording of any particular frequency as desired. The use of this device is demonstrated in Fig. 2*b*.

There are of course certain limitations in the use of the frequency analyser; e.g. it does not differentiate between a rhythm of amplitude x present for the whole epoch and a similar rhythm, of amplitude 2x present for half an epoch; nor does it convey any information on the phase relationships of the various components. Furthermore, frequency analysis assumes all waveforms to be made up of a number of sinusoidal components, and this assumption is not necessarily valid for electrophysiological mechanisms. The analyser must therefore always be interpreted in conjunction with the primary record, and must be used with full regard for its limitations.

Recording Techniques

EEGs are usually recorded with the subject lying on a couch, since this reduces the likelihood of muscle artifacts due to tension in the head and neck. Electrodes are secured to the scalp either by a head-net or by collodion, the skin having first been cleaned with alcohol. For satisfactory recording it is necessary for the skin resistance at the electrode contact to be of 5.000 ohms (Ω) or less, so that it is sometimes also necessary to rub the skin with an electrode jelly or paste.

Recordings generally last for at least 20 minutes, during which time tracings are derived from a number of different electrode combinations, so that the record finally contains a comprehensive representation of the electrical rhythms from all parts of the head, together with their interrelationships. EEGs are usually recorded whilst the eyes are closed, but observation is always made of the effects of eye opening and closure, in addition to several periods during which eyes are open.

A most important part of electroencephalography is that in which the operator deliberately alters the state of the subject, so that observation can be made of any correlated changes in the EEG, some of which may be of great value in the detection of pathology. Such a technique is known as activation, and the following are the most widely-used methods.

Hyperventilation. The subject is instructed to breathe in and out deeply for three minutes, thereby inducing a moderate alkalosis which causes a constriction of the cerebral capillaries. In some persons this may evoke abnormal electrical activity in the brain, and this will be of help in arriving at a clinical diagnosis. It is particularly valuable in cases suspected of *petit*

Fig. 3: EEG during Minor Epileptic Seizure. Female child aged 11 years; *petit mal* epilepsy. Spontaneous outburst of typical high voltage (up to 300 μv) 3 per second waves-and-spikes accompanying *petit mal* attack. The waves-and-spikes are strongest from the frontal areas, this being usual in *petit mal*. During the seizure the patient became unconscious with eyes rotated upwards, showing little movement of the body. The beginning of the seizure is indicated by an arrow.

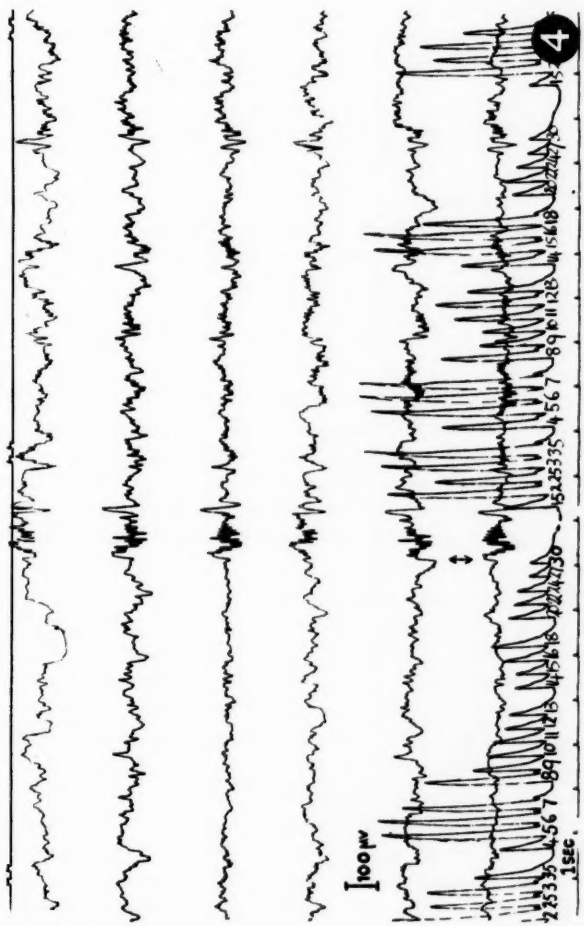


Fig. 4: EEG during Sleep. Normal female aged 27 years. Sample initially dominated by irregular medium-high voltage slow activity with frequencies from 2–8 c./sec.; at arrow there is a marked increase in faster activity, particularly 15–18 c./sec., although with no reduction in slow activity. This change represents a shift from deep to lighter sleep. Note how the adult sleeping EEG is totally different from the waking EEG (cf. Fig. 2*b*).

mal epileptic attacks, since often these are precipitated by a short period of hyperventilation and even if not overtly apparent they are immediately reflected in the EEG in the form of high voltage outbursts of the characteristic 3 per second wave-and-spike pattern (Fig. 3).

Sleep. During sleep the EEG is quite different from that in wakefulness (Fig. 4). It is also possible that in some persons, especially epileptics, significant abnormalities may appear, even when the waking patterns are quite normal. A sleeping EEG, whether natural or drug-induced, is therefore often included as part of the EEG examination. Whilst it is possible for almost any of the wide variety of epileptic abnormalities to appear during sleep, it is especially valuable for the evocation of focal spikes from the temporal regions in patients suffering from psychomotor epilepsy and temporal lobe behaviour disorders. The social significance of these two disorders is discussed in Part II of this paper.

Rhythmic Photic Stimulation (Flicker). Brief high intensity flashes of light exposed to the eyes produce observable responses in the EEGs of most persons. In 1946, Walter and his associates[34] demonstrated that under certain conditions of photic stimulation, particularly those relating to frequency, epileptics are liable to show significant abnormalities in their EEGs, and may even evince a seizure if stimulation is not terminated immediately. Flicker thus provides a simple, easily-controlled and highly satisfactory activation technique. The critical frequency range is from 3–30 flashes per second (f/s), and the stimuli are recorded by means of a photocell connected to one of the EEG channels. An example of an abnormal flicker response is shown in Fig. 5.

Metrazol. This is a convulsant drug and in many epileptics the convulsive threshold to injection of Metrazol is much lower than in normal persons. Since during such injection the EEG usually gives prior warning of an impending convulsion, this activation technique can be valuable by establishing a patient's susceptibility to seizures, in terms of his preconvulsive (EEG) threshold to Metrazol. Because the EEG can give these early indications of instability, the amount of Metrazol injected in routine examinations is usually insufficient to evoke seizure, the electroencephalographer being content to observe the EEG response to a fixed quantity, and to assess this response as normal or abnormal.

Photo-Metrazol. In 1950, Gastaut[11] reported that a combination of flicker and continuous slow injection of Metrazol is a highly sensitive activation procedure. It enables measurement of what is called the 'photomyoclonic' threshold, which is that point at which the patient displays characteristic epileptic discharges in his EEG, associated with jerks or twitches of the extremities of his body. As soon as this jerking starts, the lamp is switched off, whereupon the level of activation immediately drops to that of the Metrazol alone. Most epileptics have a very much lower photomyoclonic threshold than normal subjects, and many EEG centres

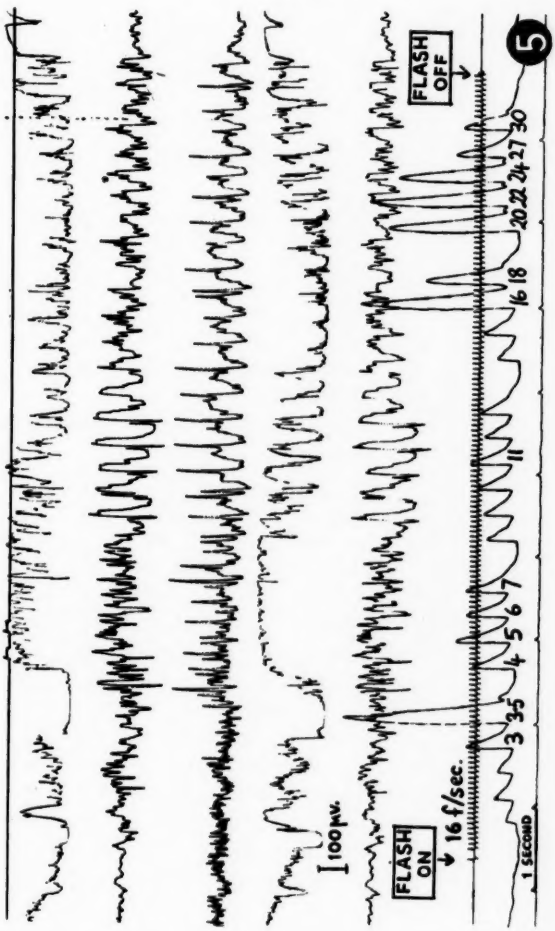


Fig. 5: Abnormal EEG Response to Flicker. Female aged 19 years; epileptic psychosis. Rapid build-up of wide-spread slow waves (3.5 c./sec.) and high voltage spikes during rhythmic photic stimulation. Such abnormal (epileptiform) discharges were often accompanied by slight myoclonic jerks from the patient. Note that one of the peaks in the spectrum is at the stimulus frequency (16 c./sec.). The prolonged slow deflections in channels 1 and 4 are due to eye movements.

maintain that photo-Metrazol activation is one of the most reliable techniques available. As with all these activation methods, it is not conclusive in the diagnosis of epilepsy, since normals, psychoneurotics, behaviour deviates, psychopaths and psychotics may possess abnormally low thresholds to such activation. This problem is at present one of the major fields of investigation among electroencephalographers, and its implications are discussed in Part II. The great importance of activation techniques in clinical electroencephalography should not, however, be underemphasized· since by their use the value of the EEG as one of the aids to final diagnosis is increased by 20–40%; indeed by application of a wide range of activation techniques it may well soon be possible to elicit abnormalities in the EEGs of nearly 100% of the epileptics[4] (the resting interseizure EEGs of 10–40% of epileptics are normal, this figure varying according to the type of epilepsy and the mean age of the group under study).

Training and Qualification of Electroencephalographers

The emergence of electroencephalography as a science in its own right was extremely rapid: from 1929 to 1934 there was considerable controversy whether the Berger rhythm existed or not, yet only 20 years later the EEG was accepted as a routine procedure in most neuropsychiatric hospitals. The British EEG Society was the first to be formed, in 1943, followed by the American in 1946. There are now numerous national EEG societies in the world, and the first international EEG Congress was held in London in 1947. At this convention an international quarterly journal was founded, called *Electroencephalography and Clinical Neurophysiology,* whilst at the second congress, the *International Federation of Electroencephalographic Societies* was formed. As these unifying movements took place, so were developed means for the examination and certification of apparatus, together with more concerted attempts at comprehensive training and qualification of personnel. This was of great importance, since before this time, potential electroencephalographers had more or less to find things out for themselves as best they could, whilst spending a few months at one or more recognized EEG centres. That this was not an unsuccessful grounding is clear from the quality of material published in EEG fields during this period, but the rapid expansion of the science, its impingement on so many other different sciences and the extent to which it is used in clinical work, made imperative the need for some more systematic approach to training. This culminated (1950)[29a] in the establishment by the American EEG Society of a Board of Qualification, which has organized an examination for candidates in the clinical application of electroencephalography. This examination is in two parts, one written and one oral, and covers electronics, apparatus, technique, neurophysiology, analysis and interpretation of records, history and literature, and all phases of electroencephalography including those aspects of

neuro-anatomy, neuropathology, neurology, neurosurgery and psychiatry that have a bearing upon the clinical application of this technique. Also given is a practical examination in the EEG laboratory with patients, as well as a review of typical records submitted by the applicant, and his interpretation of representative EEG records. The conditions of eligibility are an M.D. or Ph.D. degree and at least one year of training, or its equivalent, in electroencephalography, in addition to two years of experience.

Whilst certification as clinical electroencephalographer is not yet general practice, this development by the American EEG Society points the way to a thorough systematization of training and qualification in electroencephalography.

It will be noticed that one of the criteria of eligibility is an M.D. *or* Ph.D. degree; in other words, both medical and non-medical persons can become eligible. Some regard this as a problem. If this is so, it has been admirably resolved by Schwab [29b] in an address originally delivered in 1947, but most of which is still applicable today. The substance of his comment is as follows.

After pointing out that the early pioneers in electroencephalography were psychiatrists, physiologists, physicists, psychologists and engineers, Schwab notes that with the increasing number of clinically trained men in the field there is talk, writing and feeling that the interpretation of clinical records should be restricted to men with M.D. degrees, who have the legal responsibility and *perhaps* the clinical experience to make a diagnosis of a disease.

His conception of the main problem is that in the eyes of hospital administrators, *opinions* must be backed by appropriate signatures of M.D.'s, but *descriptive facts* may be entered by anyone at all. But if an EEG report is confined to a description, invariably the referring doctor calls for an explanation—as would the average neurologist or psychiatrist. (Schwab supports the latter statement by noting that only 10% of candidates he saw recently (1947) could state that they were familiar with EEG records, let alone interpret them.) Whilst admitting that this is surely a passing phase, he considers that the present situation is close to being deplorable, particularly since the few neurophysiologists that there are, are not interested in clinical routine. He concludes that the field of EEG is temporarily badly over-expanded, in that there are insufficient non-medical persons to supply the demand, leaving aside the M.D.'s. The future holds more promising prospects, however, since more and more neurologists and psychiatrists are becoming acquainted with electroencephalography. In the meantime, reliance must be placed on cooperative teamwork between medical and non-medical persons, until such time as the latter can be freed from the burdens of clinical routine. Schwab concludes that it seems wise that every clinical laboratory should have medical supervision, and that no final diagnosis should be made without a medical opinion on the EEG report.

All these considerations are clearly of relevance to the medico-legal aspect of electroencephalography.

Appendix I: Glossary

Alkalosis. Increased hydrogen ion concentration in the blood, i.e. a shift towards alkalinity in the acid-alkali balance of the blood. This can be due to washing out of carbon dioxide from the blood by hyperventilation (continuous rapid deep breathing).

Artifact. Any recorded potential which does not originate directly in the brain. Artifacts can be due to (a) biological potential changes; (b) defects in the recording apparatus; or (c) extraneous interference.

Cerebral Cortex. Outer or surface layer of the brain composed of grey matter. One of its main functions is the analysis, interrelation and integration of incoming sensations. Both cortical and subcortical regions (e.g. thalamus—*q.v.*) are of fundamental importance in such activities as perception, memory, learning, thought and feeling.

Cycle. The complete series of changes undergone by a wave before the sequence of changes is repeated.

Cursor. A transparent measuring device placed on the record for establishing the frequency of the various components and the phase relations of the various traces.

Electrode. The terminal of the EEG that is in physical contact with the subject.

Epoch. An arbitrary period of time during which an analysis is made.

Functional Disorder. Disorder not involving any change or disease affecting structure, as contrasted with organic disorder, e.g. a mental disorder which can be formulated in terms of disorder of behaviour without reference to organic change in any part of the nervous system. The differentiation is sometimes dubious.

Galvanometer. An instrument for measuring current by electro-magnetic action.

Ions (Lit. 'going things'). Charged portions of molecules; they may be either positively or negatively charged.

Microvolt. One millionth of a volt—the standard voltage unit in electroencephalography.

Neurone. The structural unit of the nervous system, consisting of a cell body with its processes. Also known as *nerve cell.* There are approximately 10^{10} neurones in the human brain.

Phase. The time relations of the different parts of a wave in a single record, or of a wave as recorded in several channels.

Psychopath. One who continually exhibits antisocial or asocial conduct, often of a recurrent or episodic type, which remains relatively unaffected by social, penal or medical care.

Resistance. The electrical analogue of friction, measured in ohms. Ohm's law states that for a steady condition the current through a conductor is proportional to the applied potential (voltage) and inversely proportional to the resistance.

Sinusoidal. Having the shape of a sine wave, i.e. smooth, regular and rounded.

Spectrum. Display of an analysis in a progressive order of frequencies.

Spike. A transient potential change of such short duration that when it is recorded on the EEG at standard paper speed (1.5 or 3 cm./sec.) it appears as a rising and falling vertical line, or as an extremely sharp wave when compared with an alpha wave.

Thalamus. Mass of grey matter situated near the base of the brain (i.e. subcortical) which includes a series of relay and sorting stations through which most sensory impulses must pass before they can reach the cerebral cortex (*q.v.*).

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The Clinical Criteria of Intoxication and the Chemical Analysis of the Blood and the Urine

An Investigation of 100 Cases of Motor Vehicle Drivers Charged with being Under the Influence of Alcohol

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In the Union of South Africa, during 1952, there were 48,440 road accidents involving 102,059 vehicles, viz. 63,722 motor cars, 22,654 commercial vehicles, 2,581 motor cycles, 9,883 pedal cycles and 3,219 other vehicles. As a result of these accidents 1,061 people were fatally injured, 3,857 were seriously injured and 14,312 were slightly injured. It is impossible to state in how many of these road accidents alcohol played a part, either directly or indirectly, as only those in which the driver was actually charged with being 'under the influence' are known; but it is a reasonable guess that, had thorough investigations been made into the state of each person involved, alcohol could be said to have played a part, even to the smallest degree, in 15—30%.

The diagnosis of border-line cases of being 'under the influence' has always been difficult; also, testimony in court on the clinical features alone is insufficient in the majority of cases. Thus this investigation attempts to correlate the clinical findings of drunkenness with the concentration of alcohol in the blood and the urine at the time. The work is based on the clinical and chemical features of 100 cases of drivers charged with having their faculties impaired in one way or another by alcohol, while in charge of motor vehicles.

Such a series might give chemical standards for the diagnosis of driving a motor vehicle 'while under the influence of alcohol'. Throughout this investigation 'drunkenness' included any loss of muscular co-ordination; without this no positive diagnosis was made.

Methods

I. For a positive clinical diagnosis of drunkenness, the subject had to have:

1. *Dysfunction of the motor system* as exhibited by:

(*a*) *Abnormality of gait* (indicated by (1) in the Tables). For the diagnosis of gait, the

subject walks from one side of the room to the other with his eyes open, turns, faces the examiner and then walks back with his eyes closed. This is marked as normal, slight staggering, marked staggering or failure.

(*b*) *Romberg test* (indicated by (7) in the Tables). Here the subject stands with his feet together, arms outstretched, first with his eyes open for 30 seconds and then with his eyes closed for 30 seconds. This is marked as normal, swaying with the axis in the shoulder region, swaying with the axis in the ankles, or complete failure.

(*c*) *Finger-finger test* (indicated by (8) in the Tables). The suspect with his eyes closed is asked to extend his arms and then bring the forefingers of the 2 hands together. It is marked as successful, some of the strikes miss, more than half of the strikes miss, or complete failure.

(*d*) *Picking up matches from the floor* (indicated by (9) in the Tables). Four matches are thrown to the floor and the subject is asked to pick them up one by one, placing them on an adjacent table. He may be successful, partially successful, very poor or show complete failure.

In the final assessment of abnormality of motor function (which is indicated by (1) in the Tables) all the above tests are considered as a whole.

Thus for the *positive diagnosis* of intoxication the 'suspect' must have this *abnormality of motor function* together with at least 2 of the following:

2. *Psychological abnormality* (indicated by (2) in the Tables) as exhibited by speech, behaviour, memory and orientation.
3. *Flushing of the face* (indicated by (3) in the Tables).
4. *Dilatation of the pupils* (indicated by (4) in the Tables).
5. *Odour of alcohol on the breath* (indicated by (5) in the Tables).

The degree of intoxication was classified as follows:

0 = Not intoxicated;
1 = Slightly intoxicated but given the benefit of the doubt;
2 = Intoxicated;
3 = Markedly intoxicated.

Also in the Tables:

M = Moderate drinker;
O = Occasional drinker;
H = Heavy drinker.

II. For the chemical analysis of the blood and urine the method of Kozelka and Hine[1] was adopted.

The blood was removed from an ante-cubital vein at the time of clinical examination. Sodium fluoride was used as an anticoagulant and the skin was cleaned with 1:1000 mercuric chloride solution.

When the subject was first seen he was asked to empty his bladder and this specimen of urine was discarded. Fifteen minutes later, when the blood was removed, the subject was again asked to urinate. This was the specimen used in the analysis.

Results

1. Table 1 demonstrates that the ratio of alcohol in the blood to that in the urine is 1·00 : 1·31. This compares favourably with Jetter's findings[2, 3] of 1·32.

2. Tables 1 and 2 show that an alcoholic odour was present on the breath of all the cases, but only 80% of them were classified as intoxicated.

Motor abnormality, especially that of gait (1) was present in 82% of the cases and of these 97·5% (i.e. 80) were classed as clinically intoxicated.

TABLE 1: DEMONSTRATING THE CLINICAL CRITERIA OF INTOXICATION WITH THE CONCENTRATION OF ALCOHOL IN THE BLOOD AND URINE IN 100 CASES: ALSO THE STATE OF INTOXICATION

Case No.	*Clinical Criteria of Intoxication*									*Degree of Intoxication*	*Type of Drinker*	*Concentration of Alcohol* (Blood)	*Concentration of Alcohol* (Urine)	*Ratio Blood : Urine*
	1	2	3	4	5	*6	7	8	9					
1	+	+	+	0	+	94	+	0	0	2	H	0·22	0·28	1·27
2	+	0	+	+	+	120	0	0	0	2	M	0·16	—	—
3	0	0	+	0	+	88	0	0	0	0	O	0·04	—	—
4	+	+	+	0	+	112	+	0	+	2	M	0·25	—	—
5	+	+	0	0	+	104	0	+	+	2	M	0·26	0·32	1·24
6	+	0	+	0	+	80	0	+	+	2	H	0·15	0·20	1·33
7	+	0	+	0	+	120	0	0	0	2	O	0·11	0·17	1·55
8	+	+	0	0	+	92	0	+	+	3	H	0·29	—	—
9	+	0	+	+	+	96	0	+	0	2	M	0·16	—	—
10	+	+	+	0	+	100	+	+	+	3	H	0·47†	0·53†	1·13
11	+	0	+	+	+	112	0	+	0	2	H	0·11	0·16	1·45
12	0	0	+	0	+	88	0	0	0	1	M	0·07	—	—
13	+	0	+	0	+	124	0	+	0	2	H	0·18	0·24	1·33
14	+	+	+	+	+	124	+	+	+	2	H	0·20	0·24	1·20
15	0	0	+	0	+	80	0	0	0	0	M	0·11	0·16	1·45
16	+	+	0	0	+	80	0	+	+	2	M	0·23	—	—
17	+	+	+	0	+	128	0	+	+	2	H	0·25	0·29	1·16
18	+	+	+	0	+	88	0	0	0	2	M	0·13	0·19	1·46
19	+	0	+	+	+	100	0	0	0	2	O	0·11	0·18	1·63
20	+	+	+	0	+	96	0	+	+	3	M	0·20	0·26	1·27
21	+	+	+	0	+	108	+	+	+	3	H	0·30	0·38	1·26
22	0	0	+	0	+	96	0	0	0	1	O	0·11	0·18	1·63
23	0	0	0	0	+	92	0	0	0	0	H	0·03	—	—
24	+	+	+	+	+	92	0	0	0	2	M	0·15	0·18	1·20
25	+	+	0	+	+	100	0	+	+	3	M	0·18	0·25	1·39
26	+	+	0	0	+	104	+	+	+	3	H	0·26	0·30	1·15
27	+	+	0	+	+	110	0	0	0	2	O	0·11	0·18	1·64
28	+	+	+	0	+	116	0	+	+	3	H	0·23	0·28	1·22
29	+	+	0	0	+	102	+	+	+	2	M	0·21	0·28	1·33
30	+	0	+	0	+	104	0	0	0	2	O	0·13	0·18	1·38
31	+	+	+	+	+	100	0	+	+	2	H	0·20	0·28	1·40
32	+	+	+	+	+	130	0	+	+	2	O	0·15	0·20	1·33
33	+	0	+	0	+	84	0	+	+	2	M	0·17	—	—
34	0	0	+	0	+	78	0	0	0	0	O	0·05	—	—
35	+	+	0	0	+	130	+	+	+	3	H	0·34	0·34	1·00
36	+	0	+	0	+	116	0	+	0	2	H	0·20	0·25	1·25
37	+	+	+	+	+	76	0	+	+	2	M	0·18	0·22	1·22
38	+	0	+	0	+	84	0	0	0	2	M	0·15	—	—
39	+	+	+	0	+	104	+	+	+	2	H	0·23	0·28	1·22
40	+	+	+	0	+	92	0	+	+	2	M	0·20	0·27	1·35
41	+	+	+	0	+	108	0	0	+	2	O	0·15	0·22	1·44
42	0	+	+	+	+	100	0	0	0	0	M	0·15	0·23	1·61
43	+	+	0	0	+	108	0	+	+	2	M	0·17	0·22	1·61
44	+	+	+	0	+	120	+	+	+	3	H	0·30	0·35	1·16
45	+	+	+	+	+	120	0	0	0	2	O	0·16	0·21	1·31
46	+	+	0	0	+	116	+	+	+	3	H	0·32	0·38	1·19
47	+	+	+	0	+	96	0	+	+	2	O	0·24	0·28	1·16
48	0	+	+	0	+	116	0	+	+	1	M	0·22	0·27	1·23
49	0	+	+	+	+	100	0	+	+	1	O	0·14	0·22	1·57
50	+	0	+	+	+	98	0	0	+	2	O	0·11	0·18	1·63
51	+	+	+	0	+	88	0	+	+	2	H	0·19	—	—
52	+	0	+	0	+	112	0	+	+	2	O	0·20	0·24	1·20
53	+	+	0	0	+	72	0	0	0	2	M	0·18	0·24	1·33
54	+	+	+	0	+	104	+	+	+	3	H	0·28	—	—
55	+	+	+	0	+	88	0	+	+	2	H	0·19	0·24	1·26
56	0	0	+	0	+	120	0	0	0	1	M	0·18	0·24	1·33
57	+	+	+	0	+	116	+	+	+	2	M	0·21	0·28	1·33
58	+	+	+	0	+	100	0	+	+	2	H	0·25	0·32	1·28
59	+	+	+	0	+	112	+	+	+	3	H	0·35	0·40	1·14
60	+	0	0	0	+	96	0	0	0	0	O	0·15	—	—
61	+	0	+	0	+	112	0	+	+	2	H	0·17	0·22	1·29
62	+	0	+	+	+	96	0	0	0	2	M	0·15	0·24	1·60
63	+	+	0	0	+	110	0	0	0	2	O	0·18	0·22	1·22
64	+	0	0	+	+	112	0	+	+	2	M	0·15	0·20	1·33
65	+	+	+	0	+	120	+	+	+	3	H	0·37	—	—
66	+	+	+	0	+	84	0	0	+	2	H	0·24	0·32	1·33
67	0	0	+	0	+	80	0	0	0	1	M	0·12	0·19	1·59
68	+	+	+	0	+	116	+	0	+	3	H	0·30	0·35	1·15
69	0	0	0	0	+	92	0	0	0	0	O	0·06	—	—
70	+	+	+	0	+	116	0	+	+	3	O	0·26	0·30	1·15
71	+	0	+	0	+	112	0	0	0	2	O	0·09	—	—
72	+	+	+	0	+	108	+	+	+	3	M	0·27	0·33	1·22
73	+	0	0	0	+	84	0	0	0	0	M	0·04	—	—
74	0	0	0	0	+	100	0	0	0	0	O	0·07	—	—
75	+	+	+	+	+	104	0	0	+	2	O	0·13	0·19	1·46

TABLE 1: DEMONSTRATING THE CLINICAL CRITERIA OF INTOXICATION WITH THE CONCENTRATION OF ALCOHOL IN THE BLOOD AND URINE IN 100 CASES: ALSO THE STATE OF INTOXICATION (*contd.*)

Case No.	Clinical Criteria of Intoxication									Degree of Intoxication	Type of Drinker	Concentration of Alcohol (Blood)	Concentration of Alcohol (Urine)	Ratio Blood : Urine
	1	2	3	4	5	*6	7	8	9					
76	+	+	+	0	+	104	0	+	+	2	M	0·21	0·28	1·33
77	+	0	+	+	+	88	0	0	0	2	M	0·16	—	—
78	+	+	+	+	+	108	0	+	+	2	M	0·16	0·19	1·19
79	+	+	+	0	+	120	+	+	+	3	H	0·31	0·39	1·26
80	0	0	+	0	+	108	0	0	0	0	O	0·10	0·14	1·40
81	+	0	+	0	+	124	0	+	+	2	O	0·19	0·23	1·21
82	+	+	+	+	+	120	0	0	0	2	O	0·19	0·24	1·26
83	+	0	+	0	+	92	0	0	0	2	M	0·14	—	—
84	0	0	+	0	+	96	0	0	0	0	O	0·09	—	—
85	+	+	+	0	+	120	+	+	+	2	H	0·22	0·26	1·17
86	+	0	+	+	+	88	0	0	0	2	M	0·14	0·19	1·36
87	+	+	+	+	+	112	0	+	+	2	O	0·17	0·19	1·12
88	+	+	+	0	+	124	+	+	+	3	M	0·35	0·39	1·12
89	+	0	+	0	+	120	0	0	0	2	O	0·17	0·25	1·47
90	0	0	0	+	+	90	0	0	0	0	O	0·03	—	—
91	+	+	+	+	+	104	0	+	0	2	O	0·14	0·19	1·36
92	+	+	+	0	+	92	+	+	+	3	H	0·29	0·33	1·14
93	+	+	+	+	+	76	0	+	+	3	H	0·26	0·30	1·15
94	+	+	+	0	+	60	+	+	+	3	H	0·29	0·36	1·35
95	+	0	+	+	+	108	0	0	+	2	M	0·19	0·26	1·37
96	0	0	+	+	+	116	0	0	0	0	M	0·09	—	—
97	+	+	+	0	+	108	0	+	+	2	H	0·21	0·26	1·24
98	+	+	+	0	+	84	0	+	+	2	H	0·26	0·30	1·15
99	0	0	0	0	+	76	0	0	0	0	M	0·04	—	—
100	+	+	+	0	+	108	+	+	+	3	M	0·31	0·39	1·26

Mean Ratio Blood : Urine = 1·00 : 1·31

* 6 = Pulse rate per minute.

† This case was not charged with driving under the influence of alcohol, but with being drunk while in charge of a motor vehicle. He was almost comatose.

Psychological abnormality (2), especially that of speech, was present in 60% of the cases, 57 of which (95%) were clinically drunk.

Flushed face (3) was present in 80% of the cases; 84% of these showed the clinical criteria of intoxication.

Dilated pupils (4) were present in only 29% of the cases and of these 24 (83%) were adjudged drunk.

Combined motor abnormality (1) and psychological abnormality (2) were present in 57 of the 100 cases.

3. Of the clinical criteria of intoxication, motor abnormality is the most diagnostic. Alcoholic odour on the breath is of the least importance and is only of significance in a negative sense, viz. that the person had been drinking; but this does not indicate how much he had imbibed. Dilated pupils also, are not very significant, not being present in the low or in the higher group of blood alcohol concentration. It is present in the group of 0·10—0·29% and mainly in the 0·10—0·19% group. Below 0·10% and above 0·29% it appears to be of little diagnostic value.

4. Table 2 shows 14 cases classified as sober and 6 as slightly intoxicated. (These latter 6 were given the benefit of the doubt and discharged).

Of these 20 cases:

Alcoholic odour on the breath only: 4 cases.

Alcoholic odour on the breath with psychological abnormality: 0 cases.

Alcoholic odour on the breath with flushing of the face: 8 cases.

Alcoholic odour with dilatation of the pupils: 1 case.

Alcoholic odour with psychological abnormality and flushing of the face: 1 case. (He was slightly drunk).

Alcoholic odour with flushing of the face and dilatation of the pupils: 2 cases.

Alcoholic odour with psychological abnormality, flushing of the face and dilatation of the pupils: 2 cases.

Alcoholic odour of the breath with motor abnormality: 2 cases. (Both sober).

Of these 20 cases (6 slightly intoxicated) classified as sober for legal purposes (the 6 being given the benefit of the doubt) only 2 had motor abnormality and these 2 with only one of the other clinical criteria, viz. odour of alcohol on the breath.

5. The remaining 80 cases were classified as drunk:

Motor abnormality, psychological abnormality and alcoholic odour was present in 10 cases.

Motor abnormality, flushing of the face and alcoholic odour was present in 13 cases.

Motor abnormality, dilatation of the pupils and alcoholic odour was present in 1 case.

Motor abnormality, psychological abnormality, flushing of the face and alcoholic odour was present in 33 cases.

Motor abnormality, psychological abnormality, dilatation of the pupils and alcoholic odour was present in 2 cases.

Motor abnormality, flushing of the face, dilatation of the pupils and alcoholic odour was present in 9 cases.

All the clinical criteria of intoxication were present in 12 cases.

Thus of the 82 cases with motor abnormality, 80 were classified as intoxicated.

TABLE 2: DETAILED ANALYSIS OF TABLE 1

Clinical Criteria Present	*Not Intoxicated* (0)	*Slightly Intoxicated* (1)	*Intoxicated* (2)	*Markedly Intoxicated* (3)	*Total*
5	4	0	0	0	4
15	2	0	0	0	2
25	0	0	0	0	0
35	5	3	0	0	8
45	1	0	0	0	1
125	0	0	6	4	10
135	0	0	13	0	13
145	0	0	1	0	1
235	0	1	0	0	1
245	0	0	0	0	0
345	1	1	0	0	2
1235	0	0	17	16	33
1245	0	0	1	1	2
1345	0	0	9	0	9
2345	1	1	0	0	2
12345	0	0	11	1	12
Total	14	6	58	22	100

6. Table 2 also shows that of the 100 'suspects', from a study of their clinical criteria, they fall into 3 distinct groups:

Group 1: (The Sober Division).

With alcoholic odour only: 4 cases.

Alcoholic odour and motor abnormality: 2 cases.

Alcoholic odour and flushing of the face: 8 cases.
Alcoholic odour and dilated pupils: 1 case.
Alcoholic odour, flushed faces, dilated pupils: 2 cases.
Alcoholic odour, flushed face and psychological abnormality: 1 case.
Alcoholic odour, flushed faces, dilated pupils and psychological abnormality: 2 cases.
Group 2: (Intoxicated but without Psychological Abnormality).
Motor abnormality, flushed faces, alcoholic odour: 13 cases.
Motor abnormality, dilated pupils and alcoholic odour: 1 case.
Motor abnormality, flushed faces, dilated pupils and alcoholic odour: 9 cases.
This makes a total of 23 cases in this group.

TABLE 3: FURTHER DETAILED ANALYSIS OF TABLE 2

Group	*Number*	*Number of Cases*			
		Not Intoxicated		*Intoxicated*	
		Sober (O)	*Slightly Intoxicated* (1)	*Intoxicated* (2)	*Markedly Intoxicated* (3)
1. (5, 15, 35, 45, 345, 235, 2345)	20	14	6	0	0
2. (135, 145, 1345)	23	0	0	23	0
3. (125, 1235, 1245, 12345)	57	0	0	35	22

TABLE 4: DEMONSTRATING THE DIAGNOSTIC FEATURES PRESENT AND THE MEAN ALCOHOLIC CONCENTRATION AT WHICH THEY OCCUR

Diagnostic Features	*Number of Cases*		*Mean Alcoholic Concentration*	
	Blood	*Urine*	*Blood*	*Urine*
5	4	0	0·05	—
15	2	0	0·095	—
25	0	0	—	—
35	8	5	0·094	0·183
45	1	0	0·03	—
125	10	7	0·24	0·29
135	13	9	0·167	0·22
145	1	1	0·150	0·20
235	1	1	0·22	0·27
245	0	0	—	—
345	2	1	0·10	0·18
1235	33	29	0·26	0·31
1245	2	2	0·15	0·20
1345	9	6	0·15	0·21
2345	2	2	0·14	0·22
12345	12	12	0·19	0·23
Total	100	75		

Group 3: (Intoxicated with both Motor Abnormality and Psychological Abnormality).
Motor abnormality, psychological abnormality and alcoholic odour: 10 cases.

Motor abnormality, psychological abnormality, flushed faces and alcoholic odour: 33 cases.

Motor abnormality, psychological abnormality, flushed faces, dilated pupils and alcoholic odour: 12 cases.

All the clinical criteria except flushed faces: 2 cases.

This makes a total of 57 cases intoxicated and exhibiting both psychological and motor abnormality.

7. Where the diagnostic features are as in Group 1 (5, 15, 35, 45, 345, 235, 2345), the blood alcohol concentration is always below 0·15%.

Where the diagnostic features are as in Group 2 (135, 145, 1345), the blood alcohol concentration is above 0·15 but never above 0·167%.

Where the diagnostic features are as in Group 3 (125, 1235, 1245, 12345), the blood alcohol concentration is from 0·15% to as high as 0·26%.

In the case of the urine alcohol, the mean alcohol concentration in Group 1 is below 0·20% alcohol except for the 2345 group, which is 0·22%; Group 2 is over 0·20% but less than 0·24%; and Group 3 is from 0·23% to 0·31%.

This therefore indicates that motor abnormality occurs early and can be present without psychological abnormality. Both motor and psychological abnormality occur together (with one or more other symptoms) at a later stage, and with a higher alcohol concentration.

Thus when motor abnormality is present, which is the most diagnostic sign in diagnosing drunkenness, there is always a blood alcohol level above 0·15% with the exception of the 2 cases adjudged sober, where the mean blood alcohol concentration was 0·095% and only alcoholic odour on the breath was present with the motor abnormality.

8. Table 5 indicates what levels of blood and urine alcohol to adopt for a positive diagnosis of intoxication, and confirms the fact that where the blood alcohol is above 0·15% or the urine alcohol above 0·20%, there *should* be no doubt about the diagnosis although there are exceptions. Below 0·05% is sober, and between 0·05—0·15% the matter depends on the results of the clinical examination.

TABLE 5: A DETAILED ANALYSIS OF TABLE 4

Degree of Intoxication	*Number of Cases*		*Mean Alcoholic Concentration*	
	Blood	*Urine*	*Blood*	*Urine*
0 = Sober	14	5	0·075	0·18
1 = Slightly intoxicated	6	5	0·16	0·22
2 = Intoxicated	58	47	0·18	0·24
3 = Markedly intoxicated	22	18	0·30	0·34
Total	100	75		

TABLE 6: DEMONSTRATING THE PERCENTAGE INTOXICATED IN THE VARIOUS BLOOD ALCOHOL GROUPS

Blood Alcohol Range	*No. of Cases*	*No. Intoxicated*	*Percentage Intoxicated*
0·00—0·04	5	0	0
0·05—0·09	7	1	14
0·10—0·14	16	11	69
0·15—0·19	30	27	90
0·20—0·24	18	17	94
0·25—0·29	13	13	100
0·30—0·34	7	7	100
0·35—0·39	3	3	100
0·40—0·44	0	0	—
0·45—0·49	1†	1	100

† See Footnote to Table 1.

9. Table 6 shows that the majority of drunk drivers fall into the ranges between 0·10 and 0·30%.

In the range between 0·15 and 0·19% there were only 3 sober cases, and between 0·20 and 0·24% there was only 1 sober case.

TABLE 7: DEMONSTRATING THE PERCENTAGE INTOXICATED IN THE VARIOUS URINE ALCOHOL GROUPS

Urine Alcohol Range	*No. of Cases*	*No. Intoxicated*	*Percentage Intoxicated*
0·00—0·04	0	0	—
0·05—0·09	0	0	—
0·10—0·14	1	0	0
0·15—0·19	16	13	81
0·20—0·24	20	17	85
0·25—0·29	18	17	94
0·30—0· 4	10	10	100
0·35—0·39	8	8	100
0·40—0·44	1	1	100
0·45—0·49	0†	0	—
0·50—0·54	1	1	100

† See Foot ote to Table 1.

10. Table 7 demonstrates that the majority of drunk drivers fall into the groups between 0·15—0·54% urine alcohol concentration.

11. Thus everyone with a blood alcohol over 0·24% and a urine alcohol over 0·30% was intoxicated. Therefore one feels justified in establishing 0·15% blood alcohol and 0·20% urine alcohol concentration as a definite indication of 'being under the influence', whether in occasional moderate or heavy drinkers.

12. Tables 8 and 9 show that motor abnormality and psychological abnormality are parallel to the concentration of alcohol in the blood; flushing of the face also, but to a lesser extent. Dilatation of the pupils is

only of diagnostic importance in blood alcohol concentrations between 0·10% and 0·30% and urine alcohol concentrations between 0·15% and 0·35%. Below and above these levels they are not present.

TABLE 8: THE PERCENTAGE OCCURRENCE OF THE VARIOUS CLINICAL CRITERIA FOR INTOXICATION IN THE DIFFERENT BLOOD ALCOHOL RANGES

Blood Alcohol Range	*Motor Abnormality (Gait)*			*Psychological Abnormality (Speech & Behaviour)*			*Flushing of Face*			*Dilatation of Pupils*			*Alcoholic Odour on the Breath*			*Romberg's Sign*		
	No.	*No. Intox.*	%	*No.*	*No. Intox.*	%	*No.*	*No. Intox.*	%	*No.*	*No. Intox.*	%	*No.*	*No. Intox.*	%	*No.*	*No. Intox.*	%
0·00–0·04	1	0	0	0	0	—	1	0	0	1	0	0	5	0	0	0	0	—
0·05–0·09	0	0	—	0	0	—	5	1	20	1	0	0	7	1	14	0	0	—
0·10–0·14	12	11	91	5	4	80	15	10	67	7	6	86	16	11	69	0	0	—
0·15–0·19	28	26	93	15	14	93	23	21	91	17	15	88	30	26	87	0	0	—
0·20–0·24	17	17	100	16	15	94	16	15	94	2	2	100	18	17	94	6	6	100
0·25–0·29	13	13	100	14	14	100	10	10	100	1	1	100	13	13	100	6	6	100
0·30–0·34	7	7	100	6	6	100	6	6	100	0	0	—	7	7	100	7	7	100
0·35–0·39	3	3	100	3	3	100	3	3	100	0	0	—	3	3	100	3	3	100
0·40–0·44	0	0	—	0	0	—	0	0	—	0	0	—	0	0	—	0	0	—
0·45–0·49	1†	1	100	1	1	100	1	1	100	0	0	—	1	1	100	1	1	100

† See Footnote to Table 1

TABLE 9: THE PERCENTAGE OCCURRENCE OF THE VARIOUS CLINICAL CRITERIA FOR INTOXICATION IN THE DIFFERENT URINE ALCOHOL RANGES

Urine Alcohol Range	*Motor Abnormality (Gait)*			*Psychological Abnormality (Speech & Behaviour)*			*Flushing of Face*			*Dilatation of Pupils*			*Alcoholic Odour on Breath*			*Romberg' Sign*		
	No.	*No. Intox.*	%	*No.*	*No. Intox.*	%	*No.*	*No. Intox.*	%	*No.*	*No. Intox.*	%	*No.*	*No. Intox.*	%	*No.*	*No. Intox.*	%
0·00–0·04	0	0	—	0	0	—	0	0	—	1	0	0	0	0	—	0	0	—
0·05–0·09	0	0	—	0	0	—	0	0	—	1	0	0	0	0	—	0	0	—
0·10–0·14	0	0	—	0	0	—	1	0	0	5	4	80	1	0	0	0	0	—
0·15–0·19	13	13	100	7	7	100	15	12	80	14	12	86	16	13	81	0	0	—
0·20–0·24	17	17	100	12	12	83	16	13	81	3	3	100	20	17	85	1	1	100
0·25–0·29	17	17	100	16	15	94	15	14	94	3	3	100	18	17	94	5	5	100
0·30–0·34	10	10	100	10	10	100	7	7	100	0	0	—	10	10	100	4	4	100
0·35–0·39	8	8	100	8	8	100	7	7	100	0	0	—	8	8	100	7	7	100
0·40–0·44	1	1	100	1	1	100	1	1	100	0	0	—	1	1	100	1	1	100
0·45–0·49	0	0	—	0	0	—	0	0	—	0	0	—	0	0	—	0	0	—
0·50–0·54	1†	1	100	1	1	100	1	1	100	0	0	—	1	1	100	1	1	100

† See Footnote to Table 1

13. The pulse rate is not a reliable clinical sign. Generally it is more rapid in the occasional drinkers and many a heavy drinker showed no alteration in pulse rate at all. However, in many of the subjects, but not consistently so, the drunker they were the more rapid was the pulse rate.

14. An interesting analysis is demonstrated in Table 10, where the occasional, moderate and heavy drinkers are subdivided into the various blood alcohol ranges. It is shown that the occasional drinkers get drunker with lesser concentrations of alcohol in their blood than do the heavy drinkers, the moderate drinkers occupying an intermediate position.

TABLE 10: THE NUMBER OF CASES AND THE PERCENTAGE INTOXICATED OF THE OCCASIONAL, MODERATE AND HEAVY DRINKERS IN THE DIFFERENT BLOOD ALCOHOL RANGES

Blood Alcohol Range	*Occasional Drinker*		*Moderate Drinker*		*Heavy Drinker*	
	No. of Cases	*% Intoxicated*	*No. of Cases*	*% Intoxicated*	*No. of Cases*	*% Intoxicated*
0·00—0·04	2	0	2	0	1	0
0·05—0·09	5	20	2	0	0	—
0·10—0·14	10	70	5	60	1	100
0·15—0·19	8	90	16	90	6	100
0·20—0·24	2	100	7	100	9	100
0·25—0·29	1	100	3	100	9	100
0·30—0·34	0	—	2	100	5	100
0·35—0·39	0	—	1	100	2	100
0·40—0·44	0	—	0	—	0	—
0·45—0·49	0	—	0	—	1†	100

† See Footnote to Table 1

Comments

The studies in this investigation provide an answer to the safe interpretation of the chemical tests as follows:

(*a*) Where there is less than 0·05% alcohol in the blood or equivalent amounts in the urine, most people are mildly affected by alcohol, depending on their drinking habits, and they can be presumed to be not under the influence of alcohol so far as the operation of a motor vehicle is concerned.

(*b*) Where there is between 0·05—0·15% alcohol in the blood or an equivalent amount in the urine, some drivers may be under the influence and others not. Here the results of the chemical tests must be considered with the clinical observations, to guide the Court on the question of alcoholic influence.

(*c*) Where there is 0·15% or more alcohol in the blood, or equivalent amounts in the urine, the subject should be presumed to be under the influence of liquor, as far as the operation of a motor vehicle is concerned, although a few do not stagger or exhibit thick speech until a 0·20% blood alcohol concentration is reached, with an occasional habitual drinker showing no clinical criteria of intoxication even at 0·24%.

This arrangement adequately safeguards the interests of the person who has been properly temperate in his habits.

Impairment sufficient to influence driving ability adversely is demonstrated quite clearly in the average person at alcohol concentrations of 0·09—0·11% in the blood. Thus the establishment of 0·15% as the presumption limit gives a considerable degree of grace for individual variation, which protects the most resistant or tolerant drinker and also makes allowances for the mild antidotal effect of coffee, caffeine, benzedrine and similar stimulants which might have been taken.

While these quantities of alcohol seem almost negligible, it should be realized that for the average adult it requires at least two 12-oz. bottles of beer or two 1-oz. glasses of 100 proof whisky to bring him up to the lower limit of 0·05% alcohol in the blood. To reach the presumption level of 0·15% alcohol in the blood, the average adult must consume six to eight 12-oz. bottles of beer or 6—8-oz. of whisky.

Glaister[4] states that below 0·06% blood alcohol the individual may be regarded as sober; between 0·06% and 0·12% the possibility of intoxication increases; between 0·12% and 0·18%, 50% will probably be sober while a figure higher than 0·18% will probably indicate intoxication.

The findings on clinical examination are not a direct expression of the influence of alcohol, but the resultant of two counteracting powers, viz. 'intoxication' and the 'ability to pull oneself together'. It would therefore appear that if we are to employ the best evidence available concerning the sobriety or the degree of alcoholic influence of an individual in Court actions, we must, of necessity, employ the chemical tests for intoxication to confirm the medical observations of outward signs and indications.

That there exists a marked variation in the individual's reaction to alcohol is shown by the fact that at 0·24% (a concentration almost half-way to the lethal point) an individual may show so little effects as to be judged sober by our criteria; or, on the other hand, may almost be in coma at this level. It has been demonstrated that many persons show no sign of intoxication at all at 0·15%, and in view of this it is felt advisable to direct the punitive effort of the law against the individual who, because of drinking, drives his car in an unsafe manner, rather than against those whose only offence may consist of having a blood alcohol concentration of 0·15%. In these circumstances the determination of the alcohol concentration of the blood or urine may be very valuable as supporting evidence and may be given the force of fact regarding the minimum amount of alcohol consumed, without in itself constituting *prima facie* evidence of the offence.

The concentration of alcohol in the blood is much better evidence of the degree of drunkenness than the amount of alcohol consumed. The concentration found should be presented to the Court with an interpretation about the percentage of persons likely to be drunk at that concentration. This information should be considered in relation to the other evidence in the case. The blood alcohol estimation does not provide an automatic answer to the question: 'Was the person drunk?'

If we had included in our requirements for the clinical diagnosis for alcoholic intoxication tests for judgment (which was not done), it is certain that everyone[5] with a blood alcohol concentration over 0·10% would be classified as intoxicated, whether habituated or tolerant drinkers or not. Judgment is the first body faculty to be affected by alcohol and judgment warped by alcohol is a far greater cause of road accidents than is simple clumsiness of muscular action.

Driving a motor vehicle requires alertness of mind, reasonable skill and judgment, good co-ordination in the operation of the controls, a normal reaction time, a sense of confidence and no impairment of the powers of concentration. A blood alcohol concentration of 0·15% or over will impair these faculties and contribute to the probability that the 'drinker' will be party to an accident. However, a driver must not be prosecuted for having a certain blood alcohol level, whether it is 0·05% or 0·15%, but he should be 'charged' on the conclusions arrived at after considering the results of the clinical observations, the evidence of other witnesses and in the light of the chemical tests.

Summary

1. An investigation was made of 100 cases of drivers charged with driving motor vehicles while 'under the influence of alcohol'. In all of them the blood alcohol concentration was determined, and in 75% of them the urine alcohol concentration. A clinical examination was made for the degree of intoxication in every case.

2. The subjects could be divided in to 3 groups: the sober group, the intoxicated group without psychological abnormality and the intoxicated group with psychological abnormality.

3. For a positive diagnosis of intoxication, the subject had to have abnormality or motor function as determined by the gait, the Romberg sign, the finger-finger test and the test of picking up small objects from the floor, together with at least 2 of the following criteria: psychological abnormality (as determined by speech, behaviour, memory and orientation), flushing of the face, dilatation of the pupils and an alcoholic odour on the breath.

4. Of the various clinical criteria it is demonstrated that an alcoholic odour on the breath is of the least importance as it demonstrates only that alcohol has been drunk, but not how much. It is of importance in a negative sense.

Flushing of the face is of little significance as there is a great deal of individual variation.

The pulse rate, evidence of sweating and the blood pressure do not give any definite information about the alcoholic state.

The dilatation of the pupils is valuable only where the blood alcohol concentration is between 0·10% and 0·30%, as it is absent above this figure and below 0·10%, being only present between these two limits. Psychological abnormality is of importance when it occurs in conjunction with motor abnormality. Motor abnormality is the most important of the clinical criteria of intoxication.

5. A blood alcohol percentage above 0·15% is a strong indication of intoxication in all persons. Between 0·05% and 0·15% the chemical and clinical findings must be considered together and a decision made on the basis of the two. Below 0·05% the individual may be considered sober.

I desire to record my appreciation to Prof. R. H. Mackintosh and Prof. C. Laubscher for their valuable advice and encouragement.

For my first introduction into the analytical methods of alcohol estimation I am indebted to Prof. John Glaister, his University assistant (Dr. Edgar Rentoul) and his chief technical assistant (Mr. W. J. Elsey). To them and to Dr. J. A. Imrie, of the City of Glasgow Police, I am deeply grateful for the many hours they spent discussing problems of the chemical diagnosis of alcohol with me.

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I also wish to record the pleasure and profit received from advice from Drs. Keith Simpson, Francis Camps and Donald Teare.

Finally I am appreciative of the 'statistical' advice given me by Dr. Robb of the Department of Mathematics, University of Glasgow.

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Determination of Ethyl Alcohol in Blood

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The determination of ethyl alcohol concentrations in the body has become of great medico-legal importance within the last 25 years. Because of the increasing seriousness of the 'drunk driver problem', special legislation has been enacted in many states defining and condemning 'drunk driving'.[1] It is unfair, to both accuser and accused, to place full responsibility upon the police officer or even the physician for determining the degree of intoxication of an individual. There is little argument that a reliable chemical analysis of blood furnishes one with a superior means of determining the physiologic depressant effect of alcohol.[2, 3]

Drinking excess quantities of whisky (approximately 1 qt.) within a relatively short interval can and does produce death in a direct manner by respiratory depression.[4, 5] In 1952 there were at least 165 deaths in Virginia due to poisonings, alcohol leading the list again; carbon monoxide and barbituric acid derivatives following second and third respectively.

Alcohol	68.
Carbon Monoxide	30.
Barbiturates	17.

The determination of alcohol in human tissue or fluids is by far the most frequent analysis performed by a forensic-toxicological laboratory. There are many procedures devised and proposed for this determination, most of which are reliable. However, it is important to remember that even the best procedure is no better than the proficiency and experience of the performer. Therefore most of the standard tests reported are satisfactory if performed properly. As with all tests there are small inaccuracies which usually are directly proportional to the intricacies and the number of steps required for a complete determination, and factors for correction further complicate the reliability.

Most of the methods reported are modifications of the original Nicloux reaction.[6]

$$3\,C_2H_5OH + 8\,H_2SO_4 + 2\,K_2Cr_2O_7 \longrightarrow 3\,HC_2H_3O_2 + 2\,Cr_2(SO_4)_3 + 11\,H_2O + 2\,K_2SO_4$$

(Yellow) (Green)

Bogen's modification is a simple, rapid, one-step procedure which does not require any special equipment and, if certain difficulties are overcome, is accurate; results can be duplicated to about 0.01 gm. %. Some of these difficulties are:

1. Aspiration almost always produces foam which may bubble over into the reaction tube and invalidate the test.
2. Colour standards are sometimes difficult to match especially by those with poor colour acuity.
3. Permanent colour standards are photo-sensitive and deteriorate.
4. Colour standards are prepared artificially by direct addition of alcohol to the reagent and the hue of colour thus obtained does not duplicate the actual test.

These can be overcome with the following procedure.

Reagents:

1. *Anstie*: 3.70 gm. of potassium dichromate CP are dissolved in approximately 150 ml. of distilled water. To this add with caution 280 ml. of sulphuric acid CP. Cool and then add sufficient distilled water to bring final volume to 500 ml.
2. *Anti-foam*: Mix 1 part of silicone anti-foam A (Dow Chemical Co.) with 25 parts of oil of petrolatum.
3. *Scott-Wilson*: 5 gm. mercuric cyanide CP are dissolved in 300 ml. distilled water. 90 gm. sodium hydroxide CP are dissolved in 300 ml. distilled water. 1.5 gm. silver nitrate CP are dissolved in 200 ml. distilled water. When sodium hydroxide is cool, add it to the mercuric cyanide solution, mix thoroughly and then add the silver nitrate solution and again mix thoroughly. If the solution becomes cloudy or a precipitate forms on standing, filter.
4. *Standard alcohol solution* exactly: (2% for standard stock). Add about 50 ml. of distilled water into a 100 ml. volumetric flask, add exactly 2.53 ml. of absolute ethyl alcohol by means of an accurately graduated micro burette. Keep tip near surface of water in delivering. Make this solution up to 100 ml. mark with water.
5. *Alcohol-free blood* 40 ml. (for standards).

Procedure

Arrange 2 large test tubes (approximately 25 x 210 mm.) with inlet and outlet tubes (Fig. 1). Glass joints would be desirable but rubber stoppers are satisfactory. The inlet tubes should extend nearly to the bottom of the test tube, and the outlet tubes just below the stopper. The end of the tube extending into the dichromate solution should be tapered to a fine opening or blown into a small even bulb and several small perforations made. These tubes are arranged in such a manner that a current of air may be aspirated through the specimen tube into the dichromate solution.

In the specimen tube place 4 ml. of blood,* 4 ml. of Scott-Wilson reagent, 2 ml. of water, and 1 drop of anti-foam solution. Into the second test tube add exactly 9 ml. of potassium dichromate solution. Suspend both tubes in a water bath previously brought to boiling. Connect for suction and adjust so that a slow

* Tissue distillate, spinal fluid, urine, gastric lavage etc. may also be used.

regular current of air is aspirated through the tubes. Continue heat and aspiration for 15 to 20 minutes.

Cool the dichromate solution and make the final volume up to exactly 10 ml. with the addition of water. Place small aliquot into a cuvette and read with a

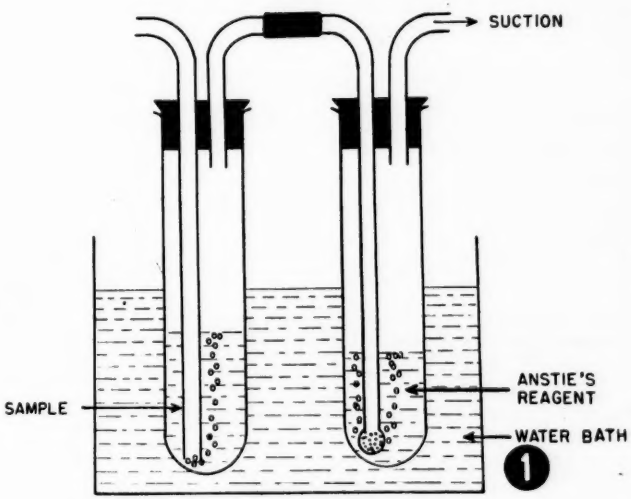

Fig. 1. *Apparatus.*

spectrophotometer at 605mμ. The report is in gm. % as determined with a standard graph (Fig. 2). The reference blank is prepared from normal blood (alcohol free). This may be used indefinitely if kept uncontaminated.

To prepare and determine the graph for alcohol standards, identical conditions of procedure must be followed. 4 ml. of normal blood is inoculated with proportional amounts of stock alcohol standards (Table 1) and is then determined exactly by procedure described above.

Table 1

Test Number	2% *Stock Alcohol Solution*	*Corresponds to gm. % Alcohol*
1	0·0 ml.	0·00 Gm. %
2	0·1	0·05
3	0·2	0·10
4	0·3	0·15
5	0·4	0·20
6	0·5	0·25
7	0·6	0·30
8	0·7	0·35

The resultant colours of reaction will be observed to range from yellow, yellow-green, to green at 0.30%. Above this, the solution will be blue-green (invalid).

Standards should be triplicated and plotted. Maximum density is at 605 mμ.

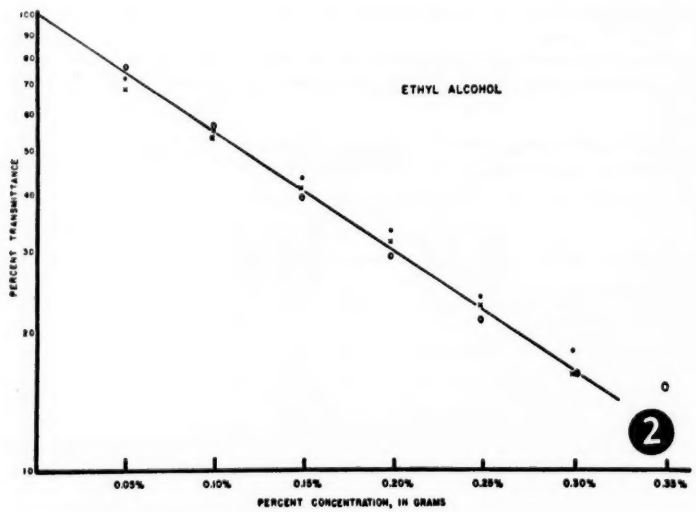

Fig. 2. This standard graph represents the most extreme curves of 3 different experiments.

Remarks

1. Approximations may be made by visual inspection with standards provided all tubes are of similar size, bore, quality, and the examiner has good colour vision. However, spectrophotometric reading is preferable. Our readings are made with the spectrophotometer (Beckman DU Universal).

2. Transition of reactant colours of mixed yellow and green follow the Beer-Lampert law up to about 0.30 gm.% at which point all the dichromate has been reduced.

3. The test can be made applicable at levels about 0.30 gm. % by:

(a) An increased concentration of potassium dichromate and the establishment of a new curve; or

(b) Performance of the test with half quantities of the specimen; 2 ml. of blood, 2 ml. of Scott-Wilson reagent, 6 ml. of water and 1 drop of anti-foam reagent. However, the resultant spectrophotometric reading is to be multiplied by 2 to convert to gm. % alcohol.

4. Speed in change of colour may be a rapid index of concentration, i.e. levels about 0.30 will produce a rapid change from yellow to green.

5. Normal blood may sometimes read as high as 0.01 gm. % alcohol.

6. The test is sensitive to about 0.01 gm. % alcohol.

7. The test can be duplicated to about 0.01 gm. %.

8. Similar to most other tests for ethyl alcohol [7-16] this procedure is not specific *per se*. Methyl alcohol, isopropyl alcohol, acetaldehyde, formaldehyde, and products of decomposition* interfere. Gettler's tests [17-19] are specific but require large quantities of specimen and are time-consuming.

9. Test specimens must be fresh or preserved.[20]

Ruling out Possible Common Interferences

Since methyl alcohol, formaldehyde, isopropyl alcohol, acetaldehyde, paraldehyde, chloroform, ether, chloral hydrate, and acetone bodies (diabetic acidosis), if present *in sufficient* quantities will interfere and yield false positive results. The above modified Bogen test was performed on a 0.05 gm. % sample of each of the above compounds in blood and then compared with ethyl alcohol standards prepared as previously described.

Table 2: Analysis of 0·05 gm. % of each and compared with Ethyl Alcohol Standards.

1. Methyl alcohol	0·09%
2. Formaldehyde	0·07% (can smell as little as 0·02% in blood).
3. Isopropyl alcohol	0·08%
4. Acetaldehyde	0·05% (can smell as little as 0·05% in blood).
5. Paraldehyde	0·01% (can smell as little as 0·01% in blood).
6. Chloroform	0·02%
7. Ether	0·00%
8. Chloral hydrate	0·00%
9. Acetone bodies (diabetic acidosis)	0·00% to 0·02%

1. *Methyl Alcohol.* From Table 2 it is observed that the presence of methyl alcohol can be a serious interference with the test and quantitation of ethyl alcohol. It is therefore necessary to perform tests and quantitation for methyl alcohol. Ozburn [21] has greatly simplified the chromotropic acid test for methyl alcohol. We find this test is sensitive to 0.00001 gm. (10 gamma) of methyl alcohol and results can be quantitated and duplicated. However, since the colour intensity produced is too great at toxic concentrations to be read with a spectrophotometer, greater dilutions are suggested.

Reagents: Potassium permanganate—dissolve 3 gm. of potassium permanganate and 15 ml. of phosphoric acid (ortho 85%) in distilled water, quantity sufficient to make 100 ml. of solution.

Sodium bisulphite—10%.

Chromotrophic acid—1, 8 dihydroxy naphthalene, 3, 6 disulfonic acid (Eastman Kodak No. 1613).

* Aldehydes, phenols, ketones, hydrogen sulphide and volatile amines etc.

Standard methyl alcohol solution—1.26 ml. absolute methyl alcohol in 100 ml. water represents a 1% standard stock solution.

Trichloracetic Acid—20%.

Technic: Place 2 ml. blood or urine* into small test tube, add while shaking, 4 ml. of trichloracetic acid. Cork and invert several times for complete mixing. Filter through an appropriate size filter, using Whatman #1. Accurately pipette 1 ml.† of clear filtrate and transfer to another test tube, to this add 2 drops of potassium permanganate and wait exactly 2 minutes. Completely decolourize excess permanganate with dropwise addition of sodium bisulphite solution (approximately 3 drops will suffice). Add a small pinch of chromotrophic acid (approximately 10 mg.) and mix thoroughly. Add 1.5 ml. sulphuric acid down the side of the test tube so as to underlay the solution. Examine interface for a purple ring. A colourless or yellow band is negative. Diffuse and completely mix this colour by swirling and allow the colour to intensify for exactly 15 minutes, at which time it is read for transmittancy in a spectrophotometer set at 570 mμ.

If methyl alcohol is present, a diffuse violet-purple colour is produced, the intensity of which will be proportional to the concentration. This can be compared with a graph of standards of methyl alcohol prepared in a similar manner as above. Large amounts of ethyl alcohol present may produce a light brown ring at the interface in the absence of methyl alcohol. If ethyl alcohol and methyl alcohol are both present, it is necessary to subtract the methyl alcohol value from the total Bogen value (using the factor 0.05% methyl alcohol produced a colour change of 0.09% on the Bogen colour scale).

Methenamine (Urotropin) and formaldehyde also give an intense purple colour. However, should there be any doubt concerning medication with methenamine, the methyl alcohol should be confirmed by the previously described test for ethyl alcohol. (Ethyl alcohol of course must be absent.)

2. *Formaldehyde.* Should there be any doubt concerning contamination of the blood or tissues with formaldehyde, as indicated by intense colour response, history, or a sharp odour recognizable even at low concentrations (0.02 gm. %); this may be detected by *omitting* the oxidation (with potassium permanganate) and decolourization (with bisulphite). Merely adding the chromotrophic acid and sulphuric acid is sufficient to develop an intense purple colour in the presence of traces of formaldehyde. If this test is positive, any further testing for methyl alcohol is not reliable. However, a presumptive estimate may sometimes be possible by estimating colour differences between the oxidized and unoxidized samples (if the formaldehyde is present only in traces).

It is a good precautionary step to check all positive methyl alcohol tests for formaldehyde, especially since it requires so little specimen (residual blood filtrate) so little time; and, of more importance, may save embarrassment of error.

3. *Isopropyl Alcohol.*

Reagents:

Denige's reagent—Suspend 2 gm. yellow mercuric oxide in 16 ml. water. Add while mixing, 8 ml. sulphuric acid. Stir and dissolve with addition of 16 ml. more water.

* Or aliquot portion of tissue steam distillate etc.

† If methyl alcohol is present in large amounts, use 0.1 ml. of clear filtrate.

Zinc sulphate—10%.
Sodium hydroxide—0.5 N.
Standard isopropyl alcohol—2% standard stock.

Technique: Place 5 ml. of blood in a 50 ml. Erlenmeyer flask. Add 5 ml. zinc sulphate solution and thoroughly mix. Slowly add 5 ml. of sodium hydroxide and mix. Filter through an appropriate sized funnel using Whatman #1. Accurately pipette 5 ml. of clear filtrate* and transfer to a calibrated centrifuge tube.† Add 10 ml. of Denige's reagent and heat in water bath for 15 minutes. A white or yellow-white precipitate forming within 15 minutes indicates the presence of isopropyl alcohol, acetone or tertiary butyl alcohol. Centrifuge and compare the quantity of the precipitate with standards similarly prepared. If ethyl alcohol and isopropyl alcohol are both present, it is necessary to subtract the isopropyl alcohol level from the total Bogen level (using the factor 0.05% isopropyl alcohol produced a colour change of 0.08% on the Bogen colour scale).

4. *Acetaldehyde.* 0.05 gm. % yields a colour change representative of 0.05 gm. % on the ethyl alcohol graph with the modified Bogen test.

To differentiate between ethyl alcohol and acetaldehyde perform Stotz'[23] test on blood filtrate or tissue distillate. The Stotz test is negative for ethyl alcohol but is positive for acetaldehyde and paraldehyde.

5. *Paraldehyde.* Paraldehyde does not interfere with ethyl alcohol when the modified Bogen test is used. Both acetaldehyde and paraldehyde can be detected and quantitated with either blood or tissue distillate by the Stotz test.[23]

6. *Chloroform.* Chloroform interference presents no problem since a 0.05 gm. % chloroform concentration in blood represents only an 0.02 gm. % coloration with the alcohol standards. Furthermore, the usual chloroform concentration present in man during the maximum anaesthesia would only be about 0.015 gm. to 0.025 gm. % in the blood and this concentration gives a negative reading with the Bogen reaction.

7. *Ether.* Ether does not present any interference with the validity of the Bogen test since a 0.05 gm. % ether concentration in the blood produces no reaction with the Bogen test. This was proven both *in vitro* and *in vivo*.

Deep anaesthesia (humans): Negative Bogen reaction.

8. *Chloral Hydrate.* Chloral hydrate in usual therapeutic or toxic levels presents no interference in blood.

Therapeutic sedation (humans): Negative Bogen reaction.

Lethal levels (as high as 40 mg. per 100 ml.): Negative Bogen reaction.

9. *Acetone Bodies* (Diabetic Acidosis). Ten hospital cases with marked acidosis (all below 10m.eq./L carbon dioxide combining power), ++++ acetone, high blood glucose (all above 500 mg. per 100 ml.) and high urine glucose.

* Or 5 ml. of tissue distillate (representing 1 gm. $\simeq$1 ml.).

† Pyrex constable protein centrifuge tubes. These are 15 ml. tubes with capillary stems graduated from 0 to 0.1 ml. in 0.005 ml. divisions. The upper section is graduated from 1 to 10 ml. These tubes are to be calibrated in an identical manner as described above using normal blood inoculated with standard isopropyl alcohol.

Without Scott's Wilson reagent: Ranged from 0.05%–0.10%.
With Scott's Wilson reagent: 0.00%–0.02% (negligible).

Table 3

Bogen	*Chromotropic Acid*	*Stotz*	*Denige's Test*
Ethyl alcohol	Methyl alcohol	Acetaldehyde	Isopropyl alcohol
Methyl alcohol	Formaldehyde	Paraldehyde	Acetone
Isopropyl alcohol	Methenamine		
Formaldehyde			
Acetaldehyde			

10. Swabbing the arm with alcohol before venipuncture should be avoided since it always poses an important question of contamination. However, a series of 25 blood specimens was taken at random from a hospital laboratory. These bloods were collected in the usual manner (alcohol swabbing of the arm), for routine blood chemistry. Alcohol determinations were performed and in each case the alcohol level was negative (below 0.01%).

This small series, although negative, does not justify the use of alcohol since one cannot rule out the possibility of error in all cases. Other substitutes for alcohol, such as mercuric chloride (1 : 2,000) etc., are preferable.

Conclusion

Bogen's method for the determination of ethyl alcohol has been modified to eliminate certain objections to reliability. Technics for the determination of methyl and isopropyl alcohol are included so that these can be determined in the presence of each other and ethyl alcohol.

Other possible interfering substances such as formaldehyde, acetaldehyde, paraldehyde, chloroform, ether and acetone bodies with alcohol are discussed from a viewpoint of increasing the reliability of ethyl alcohol determinations.

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MEDICO-LEGAL NOTES AND NEWS

British Association of Forensic Pathologists

As a result of a decision moved at the meeting of the Fellows on 28 November 1953, a meeting was arranged with the Association of Clinical Pathologists on 31 March to discuss various aspects of the status and general position of Forensic Medicine.

A Crest for the Association: At the last meeting a suggested design for a crest for the Association was incorporated in the programme.

The Private Members Bill to amend the Coroners Act of 1926 in respect of witnesses' fees and fees for post mortem examinations has had its second reading in the House of Commons and is now in the committee stage. It aims to remove the statutory fees from the 1926 Act so as to enable them to be made by order of the Home Secretary. Any increase will be borne by the Treasury and not by the local government.

The next meeting will be held in Glasgow on Saturday, 10 July, at the same time as the annual representative meeting of the B.M.A. where there is also a section of Forensic Medicine. There will be a business meeting for Fellows on the evening of Friday, 9 July, with a small informal dinner. The dinner of the Association will be on Saturday evening.

Attention has been drawn to the *British Abstracts of Medical Sciences* which include abstracts of all significant papers in Anatomy, Biochemistry, Pharmacology and Physiology.

It is a comprehensive survey of the world literature supported by Physiological, Biochemical, Anatomical, Pharmacological and Pathological Societies and other interested bodies and is being issued in 12 issues per annum at a price of £10 or printed on one side of the page only, £10 10s. 0d.

It has been suggested that Forensic Medicine might also be included and that the Association would in that event become a sponsor in company with other very reputable societies such as those named above which would involve it only in a guarantee of £10 (liability limited to this). This would entitle the Association to have a representative on the Board.

The Editorial Office is The Lister Institute, Chelsea Bridge Road, London, S.W.1.

The Seventh Meeting of the Association was held in London on Saturday, 28 November 1953, at The London Hospital Medical College (by kind permission of the Governors).

Professor Sir Sydney Smith being unable to attend, Professor John Glaister took the Chair. Forty-four members attended, including Corresponding Members and Colleagues present by invitation.

The Morning Session was opened by Professor John Glaister on *Anoxia and the Interpretations of the Post Mortem Appearances*, followed by the invited speakers:—

Professor D'Silva (Department of Physiology, London Hospital);

Dr. B. G. Lucas (Department of Surgery, University College Hospital);

Dr. J. H. Strangeways (Dublin);

Dr. H. A. Shapiro (South Africa) (Communication read);

Dr. W. H. McMenemey (Maida Vale Hospital).

A general discussion then took place.

The Afternoon Session was devoted to the presentation of short papers, members of the Association only attending.

Dr. G. Manning (Southport): *Regina* v. *Merryfield.*

Dr. F. D. Hocking (Cornwall): *Case of Neck Injuries—Suicide or Murder?*

Dr. T. Skene Keith (London): *Traffic Accident—Unusual Causes of Death.*

Dr. A. Mant (London): *Regina* v. *Whiteway.*

Dr. J. S. Faulds (Carlisle): *A Case of Head Injury.*

Dr. Harald Gormsen (Copenhagen): *Further Investigations on Alcohol Analysis of Post Mortem Blood.*

The Dinner was held at the Café Royal, Regent Street, London.

The guests of the Association were:—

Mr. W. C. Crocker—President of the Law Society.

Dr. J. C. Matheson—President of the Medico-Legal Society.

The Association was also delighted to welcome as guests two Corresponding Members, Dr. Harald Gormsen (Copenhagen) and Professor F. Thomas (Ghent).

A fuller report of the scientific papers and discussion will be available later.

REVIEWS OF BOOKS

Gradwohl's Legal Medicine

Legal Medicine. Edited by B. H. Gradwohl, M.D., Sc.D., F.A.P.H.A., Commander, M.C., U.S.N.R. (Retired). (Pp. 1093 + 222 illustrations, £8 10s.). St. Louis: C. V. Mosby Company, 1954.

Contents: 1. The History and Development of Legal Medicine (Sir Sydney Smith). 2. Special Aspects and Practical Considerations of the Medico-Legal Autopsy (Frederick D. Newbarr and Richard O. Myers). 3. Legal Authorization for Autopsy (Louis J. Regan). 4. Law Relating to Medical Practice: The Doctor's Diagnosis and Treatment of Patients (Elwyn L. Cady, Jr.). 5. Forensic Thanatology (George Strassmann). 6. Post-Mortem Changes; Vital Reactions; Fat Embolism (George Strassmann). 7. Common Causes of Unexpected Deaths (L. R. Janes). 8. Effect of High and Low Temperature, Electric Current, and Radiation (George S. Strassmann). 9. Wounds of the Head and Body and Their Interpretation (Francis E. Camps). 10. Mechanical Asphyxia (George Strassmann). 11. Pathological Findings in Poisonings (George Strassmann). 12. Recent Advances in the Treatment of Poisoning (A. W. Freireich). 13. Sudden Deaths in Infants and Children (Jesse L. Carr). 14. Septal Pneumonia as a Cause of Infant Death (M. David Orrahood). 15. Forensic Aspects of Trauma to the Central Nervous System and Its Membranes (Cyril B. Courville). 16. Rape (John J. Connor). 17. Evaluation of Evidence From the Skeleton (T. D. Stewart). 18. Dental Evidence in Identification and Criminology (David B. Scott). 19. The Examination of Hairs and Fibers (W. E. Evans). 20. Identification of Blood Stains (R. B. H. Gradwohl). 21. Blood Grouping Tests in the Determination of Nonpaternity (R. B. H. Gradwohl). 22. Paternity Proceedings and Blood Tests (Sidney B. Schatkin). 23. Examination of Seminal Fluid (R. B. H. Gradwohl). 24. Toxicology (Sidney Kaye and Leo R. Goldbaum). 25. Microscopic-Crystallographic Procedures for Identification of Drugs (William V. Eisenberg). 26. Medicolegal Aspects of Alcohol Intoxication (C. W. Muehlberger). 27. Medico-legal Problems of Workmen's Compensation (Edmund J. Thomas, Jr.). 28. Pregnancy and Abortion (George Strassman). 29. Infanticide (R. B. H. Gradwohl). 30. The Law of Abortion (Louis J. Regan). 31. Forensic Psychiatry (Val Beyer Satterfield). 32. Legal Relations of the Mentally Ill (Elwyn L. Cady, Jr.). 33. Narcoanalysis for Criminal Interrogation (James H. Matthews). 34. Lie Detection (Herbert P. Lyle). 35. The Expert Witness (Clark Sellers). 36. The Medical Witness (Ernest M. Hammes). 37. Police Laboratory Administration (Clemens R. Maise). 38. Legal Aspects of Trauma and Disease (J. L. London). 39. Official Medicolegal Investigations (R. B. H. Gradwohl).

This is a considerable and important medico-legal volume which makes quite clear that modern forensic surveys are bound to be the result of the team-work of experts. No single legal or medical specialist can to-day write authoritatively on the multifarious aspects of forensic medicine.

It is timely to have available such an excellent reference source as that provided by Dr. Gradwohl, because medico-legal practitioners may well become involved in many of these highly specialized branches of knowledge in the course of one trial or consultation.

As Dr. Gradwohl has covered many topics in which important developments or critical re-assessments have taken place in recent decades, the volume is bound to have a certain episodic construction, which is indeed no fault. The Editor was stimulated to prepare this book by the important contributions made at the annual meetings of the American Academy of Forensic Sciences, reflecting substantial additions to our knowledge in this field. As we will point out in this review, many of these same topics have also been dealt with in the first 4 issues of this *Journal*, thus confirming the extent to which major problems exercise the minds of forensic contributors at present. Dr. Gradwohl's volume should also be considered alongside another valuable contribution in this field, viz. Dr. Keith Simpson's *Modern Trends in Forensic Medicine*, published by Butterworth & Co. Ltd.

Dr. Gradwohl's collaborators have produced no re-hash of the run-of-the-mill material already covered adequately in the many competent textbooks available for undergraduate students. His co-authors write with a caution bred by a scientific knowledge of the subject; thus they avoid unwarranted dogmatism and extravagant claims which cannot be substantiated. It is obviously impracticable to comment on every chapter, but certain subjects have been selected for constructive criticism. The volume as a whole can only be commended in the highest terms.

1. *The Early Post-Mortem Interval.*

It is frequently implied that the determination of this important period (often vital for reconstructing the time of death) can be done with precision to the nearest hour. Strassman, however, stresses that certainty is impossible, even when reliance is placed on the temperature of the dead body. It is in this connexion that recent observations published by Schwarz and Heidenwolf in the *International Criminal Police Review*, 1953, No. 73, p. 339 and by Shapiro in this *Journal* (1954, Vol. 1, No. 3, p. 144) should be considered. The initial stage of 4-6 hours when the internal temperature of the dead body *does not alter appreciably* is a hitherto unappreciated fact which must govern all estimations of this interval in the recently deceased.

2. *Ante-Mortem versus Post-Mortem Injuries.* It is not generally realized that quite severe bruises can be inflicted on the recently dead and that these injuries may even have the microscopic appearances of ante-mortem injuries. This has been demonstrated admirably by Prinsloo and Gordon with very convincing photo-micrographs (S. Afr. Med. J., 1951, 26 May, p. 358). This knowledge will controvert much of the uninformed dogmatism with which evidence is given on this point in the Courts.

3. *Recognition of Asphyxia at Autopsy.* This vexed problem is discussed by several contributors. Gordon's views on the non-specificity of the visceral signs, and its non-existence as a pathological entity are gaining increasing acceptance (this *Journal*, 1953, Vol. 1, No. 2, p. 65). The suggestive clue to the diagnosis (when this is possible) may come from local signs and injuries dependent on the particular mode of mechanical obstruction to the airway, e.g. finger nail marks on the neck in throttling. The great reliance placed by some pathologists on small haemorrhages in the skin of the face, together with a bluish discoloration of that part is not always justified. Strassman states (p. 276): 'Occasionally hypostatic congestion and facial haemorrhages can be caused without preceding act of violence if the victim's head was in a prone-lying position after death'. The significance of this crucial observation cannot be overestimated.

4. *Paediatric Forensic Pathology.* One of the most important achievements in Dr. Gradwohl's volume is the new perspective his contributors have been responsible for in this field, especially in relation to sudden death in the first year of life, but particularly in the first 2-3 months.

It is clear from the careful studies reported here that many a case diagnosed as one of overlaying or as death due to asphyxia from other mechanically obstructive causes, is in fact due to a variety of pneumonia. This has been called plasma cell pneumonia but is perhaps best labelled septal pneumonia. It is apparently an overlooked and often unrecognized respiratory infection which is an important cause of sudden death in infancy, often wrongly interpreted as a crib or cot death not due to natural causes, with all the tragic consequences that may follow judicially and domestically from such a catastrophic error.

Bowden (this *Journal*, 1953, Vol. 1, No. 1, p. 19) has drawn attention to this pitfall in forensic pathology and the present admirable account makes it clear that in many cases we are probably dealing with a virus infection the manifestations of which can be established fairly readily by microscopy of the lungs. No autopsy on a case of sudden death in infancy can to-day be regarded as complete without such an examination, conducted along the lines described by Orrahood (Chapter 14). Careful autopsy studies have led to a decrease of reported cot deaths over the years (p. 352). It must be highly significant that the incidence of mechanical suffocation (restricted chiefly to the first year of life) parallels the occurrence of septal pneumonia (p. 359).

Orragood's chapter emphasizes once again that the signs in the internal organs cannot be relied on to establish mechanically produced asphyxia in infants (or in adults). The lesion of septal pneumonia is characteristic and removes many a so-called crib death from the category of accident or homicide and places it properly in that of natural causes. Bowden's conclusion (this *Journal*, 1953, Vol. 1, No. 1, p. 27) can be endorsed strongly: 'No coroner should accept a diagnosis of accidental suffocation or overlaying in the absence of a thorough post-mortem investigation'. 'Thorough' must include appropriate microscopic examination.

5. *Blood Tests.* This survey (including the problems of paternity) is a useful feature of an encyclopaedic work. The irrational behaviour of juries is well illustrated in the detailed report by Schatkin (in Chapter 22) of the Chaplin case, when the actor was declared the father of a child although he was clearly and completely exonerated by the scientific facts before the Court. 'The accused man in a paternity proceedings is defenceless. The law leaves him no defence, or, at least, makes it very difficult for him to defend himself. Science . . . offers to the accused a complete, decisive defence. A blood test

exclusion, therefore, expertly and conscientiously carried out, should not be nullified by the court, thus cheating the defendant of his only defense' (p. 569).

6. *Tests for Alcohol.* The section on breath tests for alcoholic intoxication will certainly not be endorsed generally. They have been criticized severely in many quarters and their limited value has recently been discussed in this *Journal* (1954, Vol. 1, No. 5, p. 249). The author mistakenly believes that the complicated behaviour of a human being can be diagnosed by an arbitrary test erroneously held to give a simple answer to a difficult question. The answer is not so easy as a chemical result, unless we treat the matter in a completely arbitrary fashion. This is foreign to most civilized concepts of law and justice. In so far as chemical evidence *is* reliable, e.g. from the blood and not from the breath, it must be regarded as only one link in the chain of evidence to be considered by the Court before a just conclusion can be arrived at. No single question with a 'yes' or 'no' reply can solve this difficult problem.

7. *Estimation of Age and Stature.* The assessment of age from the state of the cranial sutures and of stature from the long bones receive detailed description. Todd and Lyon's data are rightly criticized because they eliminated a considerable number of facts from their calculations, *inter alia* because of irregularities in the closure of the sutures and because they wished to 'smoothe out' their curves. Yet it is these very irregularities which are the variations of the normal (Singer, this *Journal*, 1953, Vol. 1, No. 1, p. 52).

The careful application of the tables of Trotter and Gleser dealing with the reconstruction of stature should be read alongside the critical analysis by Keen (this *Journal*, 1953, Vol. 1, No. 1, p. 46), who has shown that even with Trotter and Gleser's data an estimate based, e.g. on the length of a humerus, may give a variation in length reading between ' 5 ft. 2 in. and 5 ft. 10½ in.' This makes clear how limited the value of the guess is for the individual case.

8. *General Observations.* Although it is not possible to review every chapter in this valuable book, attention should be drawn to the admirable account of the much neglected subject of *Hairs and Fibres* provided by Chapter 19, as well as the useful and practical account of *Forensic Pyschiatry*. The chapter on *Lie Detection* is given with authority which present knowledge does not justify. (See Ames, this *Journal*, 1953, Vol. 1, No. 2, p. 94) and the practice of narco-analysis for criminal interrogation is unlikely to arouse universal enthusiasm. Outside of police quarters, many would regard such methods as undesirable techniques constituting an oblique form of the 'third degree'. On the other hand, the account of the differentiation of putrefaction gases from air (as in air embolism) by a simple autopsy table test is a valuable practical aid in the study of this difficult problem, and typifies the extremely up-to-date information to be found in this very comprehensive survey of forensic medicine.

Poisoning and Poisoners

The Power of Poison. By John Glaister, Professor of Forensic Medicine in the University of Glasgow. (Pp. 272. 18s.) London: Christopher Johnson, 1954.

Contents: 1. Establishment of Proof. 2. Poison or Disease? 3. Evidence of Poison. 4. Not Guilty or Not Proven. 5. Some Mass Poisoners. 6. Defeating the Poison Acts. 7. The Safeguard of Death Certification. 8. Dishes of Poison. 9. Methods and Motives. 10. The Case of Eugene-Marie Chantrelle. 11. The Case of Adelaide Bartlett. 12. The Oxgang Farm Case. 13. The Case of Louisa Merrifield. Bibliography. Index.

Professor Glaister's rich experience has ensured that his survey of criminal poisoning throughout the centuries is a readable account which will appeal to lay as well as professional readers. In non-technical language the author explains how poisons often simulate a natural disease process. The analysis of the problem is rendered still more difficult because there is frequently no clue to be found even at autopsy as the organs often do not reveal any changes characteristic of the common poisons used. Chemical tests have, therefore, become all-important. Consequently Professor Glaister's review traces at the same time the interesting development of modern toxicology.

Much of the material in the early part of the book is based on a detailed analysis of notorious cases involving such well-known names as Armstrong (arsenic), Chantrelle (opium), Chapman (antimony), Dr. Neill Cream (strychnine), Dr. Crippen (hyoscine), Dr. Lamson (aconite), Mrs. Florence Maybrick (arsenic), Dr. Palmer (strychnine), Dr. Pritchard (antimony, aconite and opium), the Seddons (arsenic), Dr. Smethurst (antimony) and Madeleine Smith (arsenic).

There is also some discussion of the personality of the prisoner. Lombroso's views on criminal stigmata and his theory that the criminal is predestined for crime from birth are to-day wholly unacceptable, as the author indicates; but the psychopathic nature and behaviour of many of these poisoners is insufficiently appreciated.

The three chapters which conclude the volume are among the most interesting and instructive in the whole book, especially as they recount cases whose presentation has not yet become hackneyed.

The trial of Adelaide Bartlett turned on an allegation of murder employing the administration of chloroform by mouth. This remarkable case ended, not surprisingly, in a verdict of 'Not Guilty'.

In the Oxgang Farm Case (1940), the accused was charged with murdering her husband by means of arsenic. The Scottish verdict of 'Not Proven' was returned. This case illustrates extremely well the protean manifestations that arsenical poisoning may assume.

On an earlier occasion in 1937, when the victim was possibly suffering from arsenical intoxication, the deceased had had an attack of jaundice. It will be recalled that in the Madeleine Smith case the suggestion that inorganic arsenic can cause jaundice was strongly controverted. To-day there need be less reluctance to attribute this sign to inorganic arsenic. Von Oettingen in his book on *Poisoning* (Heinemann, 1952) definitely states that jaundice may develop in some cases (*op. cit.*, p. 261). This unusual clinical sign of arsenical poisoning may well mislead the attending medical practitioner into thinking that he is dealing with a natural disease process.

The Oxgang Farm case illustrates another important confusion that may be created, viz. with scarlet fever, which was suspected at one stage in the deceased in the 1940 incident. Apparently arsenic can produce almost any kind of skin rash as well as a sore throat, with the result that even the suspicious practitioner may be put off his guard temporarily.

The final chapter is devoted to the recent (1953) case of Mrs. Louisa Merrifield, hanged for the murder of an elderly woman whose housekeeper she was. Mrs. Merrifield used yellow phosphorus. The pathological and toxicological report on the liver provided grounds for a remarkable hypothesis by Professor Webster (for the Defence) that the deceased died from liver necrosis due to natural causes just at that very moment before the first particles of phosphorus could be absorbed from the stomach into the circulation. This ingenious hypothesis found favour neither with the jury nor with the Court of Criminal Appeal. The fuller account of this case in the *Notable British Trials* series will be awaited with great interest.

Professor Glaister's volume will undoubtedly receive an appreciative welcome in forensic circles.

Heart Disease: Medico-Legal Aspects

Heart Disease and Industry, with Particular Reference to Workmen's Compensation Cases. By Meyer Texon, M.D. (Pp. 324. $7.50.) New York: Grune and Stratton, Inc., 1954.

Contents: 1. The Nature of the Study. 2. The One Hundred Cases. 3. Coronary Occlusion. References. Appendix. Index and Glossary.

Important though the relationship between injury and disease or disorder undoubtedly is, the available scientific literature which can guide forensic practitioners is meagre indeed, even though they are experts.

Some nine years ago the publishers of Dr. Texon's volume made a notable contribution available when they published Stern's *Trauma in Internal Disease*, a general statement covering most of the bodily systems. Dr. Texon's book, however, confines itself specifically to heart disease (primarily the arteriosclerotic variety). It faces up to the difficult (sometimes insoluble) problem whether disease after injury occurs *post hoc* or *propter hoc*.

There is a very comprehensive survey of the basic pathology of arteriosclerosis, with commendable emphasis on the natural course of this disease as it affects the heart. Without this fundamental knowledge it is impossible to evaluate a case involving compensation. The author's review should prove extremely helpful.

One of the main topics is the relationship of non-penetrating chest injury to coronary thrombosis. That this relationship may be causal in certain types of accident (e.g. compression of the chest by the driving wheel in traffic accidents) there is little doubt; but it is quite clear that thrombosis in these circumstances depends on a *previously* diseased artery. The clot will not form in a healthy vessel. The time of onset of the cardiac symptoms is also of crucial importance and this aspect is considered very thoroughly and instructively.

The consensus of opinion is that emotional or even physical stress does not produce atheroma or thrombosis. The blockage of the arteries of the heart is the 'end result of the arteriosclerotic process and is unrelated to external events' (p. 289). Many difficulties in interpretation arise because of 'inadequate records, individual variations, the natural course of disease, exposure, economics, compensation viewpoints, etc.' With regard to chronological relationships one author pointedly declares: 'Because toads appear after a rain, it is not necessary to assume that it has rained

toads; yet this form of reasoning fills our medical testimony concerning trauma' (p. 297).

Nevertheless, exertion may produce such a deficient blood supply to the heart muscle that a portion of it may die in the same way as if a coronary artery had been obstructed by a clot. This type of interference with the heart may be fatal or the patient may recover. The forensic as well as the diagnostic importance of this reaction is that it can be diagnosed, especially with electrocardiographic assistance.

The limitations of the investigation techniques are also stressed, since in the absence of control (ECG) records, an abnormal electrocardiogram following an industrial incident cannot be considered scientific proof of their causal relationship. The demonstration of such chronologic relationship is not proof of causal relationship ' (p. 296).

The importance of cardiac neurosis, whether superimposed on organic heart disease or not, is dealt with shortly. This section could well be extended to include the over-breathing syndrome in anxiety states with the production of the pseudo-anginal picture clinically.

A distinctive feature of Dr. Texon's book is the detailed reporting of 100 clinical cases which illustrate the manifold problems which can arise and the ways in which the situation can be assessed. These case reports are an attractive feature of a very well-documented treatise which has earned praise from no less an authority than Prof. Samuel A. Levine, of the Harvard Medical School. In a Foreword Professor Levine states: 'There is reason to hope that this volume may well prove to be a turning point in a healthy movement to clarify the medico-legal aspects of heart disease. It should be of great value to both the medical and legal professions'.

Mental Defect and Capital Punishment

Trial of John Thomas Straffen. Edited by Letitia Fairfield, *C.B.E.*, M.D. and Eric P. Fullbrook. (Pp. 298 + xiii, with photographs. 15s.) London: Wm. Hodge and Company Limited, 1954.

Contents: 1. First Trial, First Day—Monday 21st July 1952. 2. Second Day—Tuesday, 22nd July 1952. 3. Second Trial, First Day—Tuesday, 22nd July 1952. 4. Second Day—Wednesday, 23rd July 1952. 5. Third Day—Thursday, 24th July 1952. 6. Appendices.

The case of Thomas Straffen poses the difficult question whether a murderer who is mentally abnormal to a gross degree, emotionally unstable and with an I.Q. of 58, should be hanged.

He could appreciate right and wrong, and knew what he was doing (within the limits of a limited intelligence). He was therefore in the situation of a 9½-year-old child with adult impulses who could not reasonably fall within the confines prescribed by the M'Naghten rules. These rules define responsibility only in the full sense, and are very clear cut. They make no provision for a diminished responsibility such as might reasonably be attributed to a mentally defective adult with a mental age of 9½ years.

This doctrine of diminished responsibility has crystallized in Scottish practice over the last 80 years and has profoundly influenced Court decisions. The Editors point out in their *Foreword* that during 1900-1949, ' of 494 persons proceeded against for murder, 54 were convicted of culpable homicide after the defence had been one of insanity or diminished responsibility '.

The concept of diminished responsibility has also been invoked in cases diagnosed as psychopathic; though here it would in general be much more difficult to apply the doctrine than in the case of persons who are severely mentally defective.

Straffen's case was remarkable in other ways. On a previous occasion he had been found unfit to plead when arraigned on a charge of murdering Cicely Batstone, aged 9 years. He had not interfered with her sexually. He admitted to the murder of another little girl, and was committed to Broadmoor as he was found insane on arraignment. He alleged that he had committed the murders ' to spite the police '.

Within 3 hours of his escape from Broadmoor a third child was murdered, Straffen being recaptured within 245 minutes of his escape. On this occasion, however, Straffen (although legally insane only 8 months earlier on a similar charge) was charged as a sane person (though he was still an inmate of Broadmoor). This left the Defence with the difficult task (and in the event they were unsuccessful) of establishing his insanity within the meaning of the M'Naghten rules.

This *volte face* in the medical evidence is as remarkable as any other feature of the case. While it can be understood that a mentally defective person may not be insane (i.e. he suffers from an amentia as opposed to a dementia) the prior certification as insane remains difficult to follow as the accused's mental defect had not altered and was regarded as incurable. Electrical discharges recorded from his brain revealed an abnormally functioning organ, acquired rather than constitutional, and involving particularly

those parts considered to be in control of the higher mental processes (pp. 167, 193.)

The verbatim record of the medical evidence is a valuable feature of this report of the *Trial*, and demonstrates clearly how often the medical practitioner is at odds with his legal colleagues. The difference in standpoint was well brought out in Mr. Justice Cassel's charge to the jury: 'If you are satisfied . . . that it was this man's hand that brought that young girl's life to an end . . . your verdict would be one of guilty of murder . . . if that is your conclusion . . . ask yourselves whether . . . he was insane within the meaning of the criminal laws: not that he was feeble-minded; not that he had a lack of moral sense; not that he had no feeling for his victim or her relatives; not that he had no remorse; not that he may be weak in his judgment; not that he fails to appreciate the consequences of his act, but was he insane through a defect of reason caused by disease of the mind, so that either he did not know the nature and quality of his act, or if he did know it, he did not know it was wrong.' (P. 262.)

The jury found him guilty, Straffen was sentenced to death, his appeal was unsuccessful but he was reprieved on the recommendation of the Home Secretary.

This sordid series of murders illustrates once again the adequacy with which the legal machine functions but reveals what many consider a wholly inadequate set of criteria by which such crimes must be judged. The measure of reform, however, lies not with the Courts but with the legislature, which is the proper place to consider whether any change in the trial of such cases is necessary. The Scottish example certainly has much to commend it.

CORRESPONDENCE

Lunge's Test for Spent Gun Powder Grains

To the Editor: I have read with interest the article on the *Detection of Spent Gun Powder Grains* in your *Journal* (1954, Vol. 1, No. 4). While in full agreement with the criticisms of the Lunge test for nitrates for the purpose outlined in the article, there are one or two points to which I should like to draw attention.

I have always maintained that this test, used for this particular purpose, is open to objection and my reasons have been precisely those mentioned in the above article. It was, therefore, somewhat surprising to learn that a 'Government Analyst' had given evidence in favour of the test. In the second case mentioned in the same year evidence was given by me personally in Johannesburg and the fallacies inherent in the test were pointed out. The test, in effect, is for oxidizing materials and by no means specific for nitrates. It is difficult for me to reconcile the two statements in the article.

I must also point out that the α-naphthylamine test, or one of its modifications, is specific for nitrites and *not* for nitrates. It is difficult to see how this particular test could be applied in the normal way in practice to a paraffin cast of the suspected hand. It might be possible to apply a pad, suitably impregnated with the necessary reagents, to the hand. If nitrites were present they might possibly react to form spots on the pad. The presence of nitrites, if proved, would be more suggestive than the presence of nitrates.

Phenol disulphonic acid is usually employed as a specific test for nitrates but any nitrites present must first be destroyed before applying this test. This would not be easy under the particular conditions involved where the grains are more or less embedded in the surface of the skin.

I agree fully with the statement that the Lunge test for spent gun powder grains requires considerable improvement before any reliance can be placed upon the results obtained in the special circumstances under consideration. The problem requires much further detailed examination and the test should be carried out by fully qualified and competent chemists. The author of the above article is to be commended for drawing attention to the contradictions associated with the present test.

Government Chemical Laboratories,
Johannesburg.
27 July 1954.

P. R. v. d. R. Copeman.

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